Case 2:20-cv-00237-JCN	Document 137-14	Filed 06/13/24	Page 1 of 138	PageID #:
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			EXHIBI	T1

UNITED STATES DISTRICT COURT DISTRICT OF MAINE

ED FRIEDMAN)	
)	
)	
Plaintiff,)	
)	CIVIL ACTION
v.)	
)	Case No. 20-cv-00237-JDL
CENTRAL MAINE POWER)	
COMPANY,)	
)	
Defendant.)	

NOTICE TO TAKE ORAL DEPOSITION OF DAVID O. CARPENTER, M.D.

TO: David O. Carpenter, M.D.

c/o William Most, Esq. Law Offices of William Most 201 St. Charles Avenue New Orleans, LA 70170 c/o Bruce M. Merrill, Esq. 225 Commercial Street, Suite 501 Portland, ME 04101

Please take notice that on Thursday, January 13, 2022, via the online platform Zoom, Defendant in the above-entitled action will take the deposition of David O. Carpenter, M.D., beginning at 10:00 a.m., upon oral examination pursuant to the Federal Rules of Civil Procedure, before The Reporting Group, or some other officer duly authorized by law to administer oaths, and will be recorded stenographically.

The deponent is requested to produce for inspection and copying at the deposition the following documents:

- 1. A current curriculum vitae;
- 2. Any and all documents reviewed, referred to, or relied upon by the deponent in forming his opinion in regards to the above-captioned matter;

- 3. Any and all documents reviewed, referred to, or relied upon by the deponent in forming the opinions to which he testified before the Maine Public Utilities Commission in the case bearing Docket No. 2011-262.
- 4. All documents reflecting communications between the deponent and Ed Friedman or his counsel during the pendency of the Maine Public Utilities Commission case bearing Docket No. 2011-262.
- 5. Any and all documents including but not limited to any reports, prepared or created by the deponent, in regards to the above-captioned matter.
 - 6. A list of all publications authored by the deponent within the past ten years.
- 7. A list of all cases in which the deponent has testified as an expert witness within the past ten years.
- All documents pertaining to any charges or payments for the services of the 8. deponent as an expert witness in this case.

You are invited to attend and cross-examine.

Please provide The Reporting Group with your contact information and email address so that an email invitation can be sent to you and/or your representatives a link to Zoom to participate in this deposition. The Reporting Group can be reached at (207) 281-4230 or thereportinggroupmaine@gmail.com.

Dated at Portland, Maine this 5th day of January, 2022.

Christopher C. Taintor, Esq.

Russell B. Pierce, Jr., Esq.

Attorneys for Defendant

Central Maine Power

NORMAN, HANSON & DeTROY Two Canal Plaza, P.O. Box 4600 Portland, Maine 04112-4600 (207) 774-7000 ctaintor@nhdlaw.com

EXHIBIT 2

CURRICULUM VITAE

Name:

David O. Carpenter

Home Address:

2749 Old State Road

Schenectady, New York 12303

Positions Held:

Director, Institute for Health and the Environment

University at Albany

Professor, Environmental Health Sciences School of Public Health, University at Albany 5 University Place, A217, Rensselaer, NY 12144

Honorary Professor

Queensland Children's Medical Research Institute

University of Queensland Brisbane, Australia

Education:

1959

B.A., Harvard College, Cambridge, MA

1964

M.D., Harvard Medical School, Boston, MA

Positions Held:

York

9/61-6/62	Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
7/64-6/65	Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
7/65-2/73	Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
2/73-3/80	Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
3/80-9/85	Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
9/85-1/98	Dean, School of Public Health, University at Albany
9/85-Pres.	Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
9/85-7/98	Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
1/98-1/05	Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
2001-Pres.	Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
2005-Pres.	Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New

Editor-in-Chief: Cellular and Molecular Neurobiology, 1981 – 1987
Editor-in Chief: Reviews on Environmental Health 2012-present

Editor-in-Chief: Journal of Local and Global Health Sciences 2012-present Cellular and Molecular Neurobiology, 1987 - Present

Editorial Boards: Journal of Public Health Management and Practice, 1995 - 2002

International Journal of Occupational Medicine & Environmental Health

1996 - Present

Journal of Alzheimer's Disease - Associate Editor, 2007-2009

Reviews in Environmental Health; 2008-2012

International Archives of Occupational and Environmental Health; 2009-present.

Journal of Environmental and Public Health, 2009-present.

Environmental Health Perspectives, 2010-present

Global Health Perspective, 2012-present

National and International Committees:

1978, 1981	Physiology Study Section (Ad hoc member)
1979-1985	NIH International Fellowship Study Section
1974-1981	Member, Steering Committee of the Section on the Nervous System, American
	Physiological Society (Chairman of the Committee, 9/76-4/80)
1981-1989	Member, USA National Committee for the International Brain Research Organization
1985-1986	Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
1986-1987	Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
1987-1989	Member, Science Advisory Council of the American Paralysis Association
1987-1990	Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
1985-1993	Committee #79, National Council on Radiation Protection and Measurements
1986-1997	Member, Legislative and Education Committees, Association of Schools of Public Health
1989-1994	Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
1994, 1995	Federation of American Societies for Experimental Biology Consensus Conference on FY
1004, 1000	1995 Federal Research Funding
1994-1997	Member, Legislative Committee of the Association of Schools of Public Health
1997	Member, Executive Committee of the Association of Schools of Public Health
1997-2000	National Advisory Environmental Health Sciences Council of the National Institutes of Health
1998-Pres.	Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
2000-Pres.	Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
2001-2008	United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
2002-2003	Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
2003-2008	Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
2003-Pres.	Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
2004-Pres.	Member, Blue Ocean Institute Curriculum Advisory Board.
2007-2011	Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.

State and Local Committees:

1980-1987	Executive Secretary, New York State Power Lines Project
1985-1989	Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
1986-1989	Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
1991-1992	Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
1991-1992	Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
1991-1992	Member, State Task Force for the Reform of Middle Level Education in NY State
1992-1993	Member, State Needs Task Force on Health Care and Education
1987-1998	Delegate-at-Large, New York State Public Health Association
1991-1995	Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis
	Association
1994	Chair, Council of Deans, University at Albany, SUNY
1997-2008.	Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
2000-Pres.	Member, Board of Directors, Healthy Schools Network, Inc.
2000-2003	Member, Medical Advisory Board, Hepatitis C Coalition, New York
2000-2004	Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
2001-2008	Member, Board of Directors, Environmental Advocates of New York
2004-2007	Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
2005-Pres.	Member, Schooling Chefs Curriculum Advisory Board
2005-Pres.	Member, Advisory Board, Healthy Child Healthy World
2005-2008	Member, Board of Directors, Citizens Environmental Coalition
2006-2009	Member, Board of Directors, Marine Environmental Research Institute
2007-2009	Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

1959	B.A. awarded <u>magna cum laude</u> . Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, <u>Ambystoma tigrinum</u> " (Thesis advisor, Professor George Wald)
	Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded <u>cum laude</u> for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu
1987	University, Fukuoka, Japan, for a period of three months each
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies

	in environmental health.
2001	Awarded 2001 Academic Laureate from the University at Albany Foundation.
2010	Awarded the Albion O. Bernstein, M.D. Award in recognition of an outstanding contribution to public health and the prevention of disease though lifelong research of environmental health hazards and for limitless devotion to medical education by the Medical Society of the State of New York.
2011	Awarded the Rodney Wylie Eminent Visiting Fellowship 2011 at the University of Queensland, Brisbane, Australia for a period of four weeks.
Federal Gra	nts Held: (Principal Investigator Only)
1980-1983	United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs", \$76,847 total direct costs.
1982-1988	National Institute of Health, "Mechanisms of Desensitization at Central Synapses", \$464,786 total direct costs.
1984-1986	Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogse, \$330,504 total direct costs.
1986-1996	National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct costs.
1989-1993	National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total direct costs
1990-1995	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O. Carpenter, P.I. \$5,783,419 total direct costs.
1995-2001	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. ACentral/Eastern European Environ/Occup Training Program@, D.O. Carpenter, P.I. \$657,520 total costs.
1995-2001	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total direct costs.
1998-1999	Environmental Protection Agency, Alndoor Air Risk at Akwesasne - Pilot Project@, D.O. Carpenter, P.I. \$9,996 total costs.
2000-2002	Association Liaison Office for University Cooperation in Development, ACooperative Program in Environmental Health between the Institute of Public Health at Makerere University, Kampala, Uganda and the School of Public Health, University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
2001-2007	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. AMultidisciplinary Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
2006-2011	Pakistan-US Science and Technology Cooperative Program (US National Academy of Sciences). "Association of particulate matter with daily morbidity in an urban population," D.O. Carpenter, P.I., \$391,104 total costs.
2009-2013	Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2:
	Environmental contaminants and reproductive health of Akwesasne Mohawk women.

\$387,825 for year 1. D.O. Carpenter, Co-Pl.

- 2010-2013 Department of the Army, "Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, "Drinking Water Supply, Sanitation, and Hygiene Promotion: Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda". D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1RO1ES019620), "Protecting the health of future generations: Assessing and preventing exposures." PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2.354.871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson's Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, <u>The Public Radio Health Show</u> (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001.

Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

- 1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. <u>Experientia</u>, 18:337, 1962.
- 2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. <u>Experientia</u>, 18:450, 1962.
- 3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. <u>Acta Physiol. Scand.</u>, 59:126-142.
- 4. Carpenter, D.O., Engberg, i., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. <u>Acta Physiol. Scand.</u>, 59:424-437, 1963.
- 5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. <u>Acta Physiol. Scand.</u>, 63:103-110, 1965
- 6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibitibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.

- 7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. <u>J. Neurophysiol.</u>, 28:560-580, 1965.
- 8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.
- 9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. <u>J. Neurophysiol.</u>, 28:958-965, 1965.
- 10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch. Ital. Biol., 104:73-85, 1966.
- 11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. <u>J. Neurophysiol.</u>, 29:353-368, 1966.
- 12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in Aplysia neurons. J. Gen. Physiol., 50:1469-1484, 1967.
- 13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. <u>Pharmacol.</u>, 6A:351-364, 1968.
- 14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na⁺ pump to membrane potential in <u>Aplysia</u> neurons. <u>J. Gen. Physiol.</u>, 52:1-21, 1968.
- 15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.
- 16. Carpenter, D.O. Membrane potential produced directly by the Na⁺ pump in <u>Aplysia</u> neurons. <u>Comp.</u> Biochem. Physiol., 35:371-385, 1970.
- 17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of <u>Aplysia</u> neurons upon Na⁺ and Ca⁺⁺. <u>J. Cell. Physiol.</u>, 75:121-127, 1970.
- Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepin-ephrine in the presence of tetrodotoxin. <u>J. Pharmacol. Exp. Therap.</u>, 73:416-421, 1970.
- 19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.
- 20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of <u>Aplysia</u> neurons and squid axon as determined by a new technique. <u>Intl. J. Neurosci.</u>, 2:35-48, 1971.
- 21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in <u>Aplysia</u> nervous and non-nervous tissues. <u>Int. J. Neurosci.</u>, 2:49-56, 1971.
- 22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of <u>Aplysia</u> neuron somata. <u>Science</u>, 176:1329-1331, 1972.
- 23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. <u>Science</u>, 179:1336-1338, 1973.
- 24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. <u>J. Physiol. (Lond.)</u>, 229:471-493, 1973.
- 25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. <u>Aplysia</u> acetylcholine receptors: Blockage by and binding of α-bungarotoxin. <u>Brain Res.</u>, 72:225-240, 1974.
- 26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. <u>Brain Res.</u>, 73:156-160, 1974.
- 27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in <u>Aplysia</u> suggests neurotransmitter function. <u>Science</u>, 185:364-365, 1974.

- 28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of <u>Aplysia</u> neurons. <u>J. Cell. Physiol.</u>, 84:463-472, 1974.
- 29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of <u>Aplysia</u>. <u>Proc. Natl. Acad. Sci.</u> (USA), 71:4662-4665, 1975.
- 30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on <u>Aplysia</u> neurons mediate hyperpolarization by increasing membrane conductance. <u>Nature</u>, 252:483-485, 1974.
- 31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. <u>J. Neurobiol.</u>, 38:601-612, 1975.
- 32. Carpenter, D.O. and Gaubatz, G.L. H₁ and H₂ histamine receptors on <u>Aplysia</u> neurons. <u>Nature</u>, 254:343-344, 1975.
- 33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal restivity of giant axons of squid and Myxicola. J.Gen. Physiol., 66:139-148, 1975.
- 34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of Aplysia. Comp. Biochem. Physiol., 52C:23-26, 1975.
- 35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents in vitro. <u>Pflugers Arch.</u>, 359:349-356, 1975.
- 36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on <u>Aplysia</u> neurons. Nature, 258:751-754, 1975.
- 37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on <u>Aplysia</u> neurons. <u>Science</u>, 192:806-809, 1976.
- 38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. Biophys. J., 16:991-1001, 1976.
- Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α-bungarotoxin with <u>Aplysia</u> acetylcholine receptors. <u>Mol. Pharmacol.</u>, 12:999-1006, 1976.
- 40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in <u>Aplysia</u>. <u>Science</u>, 195:1004-1006, 1977.
- 41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in <u>Aplysia</u> neurons. <u>J. Neurobiol.</u>, 8:119-132, 1977.
- 42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on <u>Aplysia</u> neurons. <u>Life Sci.</u>, 20:1441-1448, 1977.
- 43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. <u>J. Electrophysiol. Tech.</u>, 6:34-41, 1977.
- 44. Yarowsky, P.J. and Carpenter, D.O. Receptors for gamma-aminobutyric acid (GABA) on <u>Aplysia</u> neurons. <u>Brain Res.</u>, 144:75-94, 1978.
- 45. Carpenter, D.O., Gaubatz, G., Willis, J.A. and Severance, R. Effects of irradiation of <u>Aplysia</u> pacemaker neurons with 20 MeV electrons. <u>Rad. Res.</u>, 76:32-47, 1978.
- 46. Yarowsky, P.J. and Carpenter, D.O. A comparison of similar ionic responses to gamma-aminobutyric acid and acetylcholine. <u>J. Neurophysiol.</u>, 41:531-541, 1978.
- 47. Blum, B., Auker, C.R. and Carpenter, D.O. A head holder and stereotaxic device for the rattlesnake. <u>Brain Res. Bull.</u>, 3:271-274, 1978.

- 48. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Dopamine-induced muscle contractions and modulation of neuromuscular transmission in <u>Aplysia</u>. <u>Brain Res.</u>, 157:167-172, 1978.
- 49. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Evidence for identified dopamine motor neurons to the gill of <u>Aplysia</u>. <u>Neurosci. Lett.</u>, 10:275-280, 1978.
- 50. Kebabian, P.R., Kebabian, J.W. and Carpenter, D.O. Regulation of cyclic AMP in heart and gill of <u>Aplysia</u> by the putative neurotransmitters, dopamine and serotonin. <u>Life Sci.</u>, 24:1757-1764, 1979.
- 51. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Res. Bull., 4:149-152, 1979.
- 52. Pellmar, T.C. and Carpenter, D.O. Voltage-dependent calcium current induced by serotonin. Nature, 277:483-484, 1979.
- 53. Ruben, P.C., Swann, J.W. and Carpenter, D.O. Neurotransmitter receptors on gill muscle fibers and the gill peripheral nerve plexus in <u>Aplysia</u>. <u>Canad. J. Physiol. Pharmacol.</u>, 57:1088-1097, 1979.
- 54. Pellmar, T.C. and Carpenter, D.O. Serotonin induces a voltage-sensitive calcium current in neurons of Aplysia californica. J. Neurophysiol., 44:423-439, 1980.
- 55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthamol., 89:641-646, 1980.
- 56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homegenates by high-performance liquid chromatography. <u>Neurochem. Res.</u>, 5:1089-1096, 1980.
- 57. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Thyrotropin-releasing hormone has multiple actions in cortex. <u>Brain Res.</u>, 194:244-248, 1980.
- 58. Meszler, R.M., Auker, C.R. and Carpenter, D.O. Fine structure and organization of the infrared receptor relay, the lateral descending nucleus of the trigeminal nerve in pit vipers. <u>J. Comp. Neurol.</u>, 196:571-584, 1981.
- 59. Auker, C.R., Parver, L.M., Doyle, T. and Carpenter, D.O. Choroidal blood flow: I. Ocular tissue temperature as a measure of flow. <u>Arch. Opthal.</u>, 100:1323-1326, 1982.
- 60. Parver, L.M., Auker, C., Carpenter, D.O. and Doyle, T. Choroidal blood flow: II. Reflexive control in the monkey. <u>Arch. Opthal.</u>, 100:1327-1330. 1982.
- 61. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Lateral olfactory tract transmitter: Glutamate, aspartate or neither? <u>Cell. Mol. Neurobiol.</u>, 1:115-120, 1981.
- 62. Scappaticci, K.A., Dretchen, K.L., Carpenter, D.O. and Pellmar, T.C. Effects of furosemide on neural mechanisms in <u>Aplysia</u>. <u>J. Neurobiol.</u>, 12:329-341, 1981.
- 63. Pellmar, T.C. and Carpenter, D.O. Cyclic AMP induces a voltage-dependent current in neurons of Aplysia californica. Neurosci. Lett., 22:151-157, 1981.
- 64. Parver, L., Auker, C. and Carpenter, D.O. Stabilization of macular temperature: The stabilizing effect of the choroidal circulation on the temperature environment of the macula. Retina, 2:117-120, 1982.
- 65. Green, R.W. and Carpenter, D.O. Biphasic responses to acetylcholine in mammalian reticulospinal neurons. Cell. Molec. Neurobiol., 1:401-405, 1981.
- 66. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Pharmacologic sensitivity of amino acid responses and synaptic activation of <u>in vitro</u> prepyriform neurons. <u>J. Neurophysiol.</u>, 48:1289-1301, 1982.

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- 17. Bolte G, Kohlhuber M, Carpenter DO and Tamburlini G Environmental inequalities among children and adolescents in Europe. Report prepared and submitted to the World Health Organization, 2009.
- 18. Toxins and the Brain. PSR's Environmental Health Policy Institute, 9 April 2012

PRE-FILED TESTIMONY OF DAVID O. CARPENTER, M.D. MPUC Docket No. 2011-00262

1	Q.	Please state your name and business address.
2	A.	My name is David O. Carpenter. My business address is:
3 4 5 6		Institute for Health and the Environment University at Albany Five University Place, Room A217 Rensselaer, NY 12144-3456
7	Q.	Briefly state your occupation, educational background and current
8		employment.
9	A.	I am a public health physician and professor, with a medical degree from Harvard
10		Medical School. I have held various positions in the public health field. My
11		current title is Director of the Institute for Health and the Environment at the
12		University at Albany and Professor of Environmental Health Sciences within the
13		School of Public Health. In addition I am an Honorary Professor, Queensland
14		Children's Medical Research Unit, University of Queensland, Brisbane, Australia.
15		Formerly, I was the Director of the Wadsworth Center for Laboratories and
16		Research of the New York State Department of Health and the Dean of the School
17		of Public Health at the University of Albany, while remaining employed by the
18		New York State Department of Health. I assumed my current position in 1998.
19		I served as the Executive Secretary to the New York State Powerlines
20		Project in the 1980s, a program of research that showed that children living in
21		homes with elevated magnetic fields coming from powerlines suffered from an

1		elevated risk of developing leukemia, and that electromagnetic field (EMF)
2		exposure altered a variety of responses studied in animals and in cellular systems.
3		After this, I became the spokesperson on EMF issues for New York during the
4		time of my employment in the Department of Health.
5		Attached as Exhibit A is my curriculum vitae.
6	Q.	Are you a member of any professional organizations?
7	A.	I participate in many international, national, state and local organizations and
8		committees as listed in my curriculum vitae along with the Honors, Awards, and
9		Fellowships I have received.
10	Q.	Have you authored any papers or journal articles?
11	A.	I have authored over 350 major publications in peer-reviewed scientific journals,
12		have edited five books and have numerous other publications as listed in my
13		curriculum vitae.
14	Q.	Briefly describe your work and experience related to the study of health risks
15		related to electromagnetic fields and radio frequency waves in the 30 MHz to
16		300 GHz range ("RF"). Identify any studies or published writings on the
17		subject.
18		I have published several reviews and have edited two books on the Biologic
19		Effects of Electric and Magnetic Fields. I am also a Co-Editor and a Contributing
20		Author of the BioInitiative Report: A Rationale for a Biologically-based Public
21		Exposure Standard for Electromagnetic Fields (ELF and RF)
22		www.bioinitative.org. This report was first published in 2007, and has just now

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been updated in 2012. The *BioInitiative Report* documents bioeffects, adverse health effects and public health conclusions about impacts of electromagnetic radiation (electromagnetic fields including extremely-low frequency ELF-EMF and radiofrequency /microwave or RF-EMF fields). I will refer to specific sections of the report where appropriate but I also reference the entire report as a comprehensive and up-to-date review of the scientific information on this subject.

In 2009, I was invited to present to the President's Cancer Panel on the subject of power line and radiofrequency fields and cancer, and have also testified on this issue before the United States House of Representatives.

- Are you familiar with peer-reviewed studies addressing the biological effects of exposure to low-level RF, and their potential health effects?
- A. There are many peer-reviewed studies reporting biological effects and health risks related to low-level RF exposure. A comprehensive listing of these publications is found in the *Bioinitiative Report*, which includes both positive and negative research studies. In this testimony, I will not list peer-reviewed publications dated prior to 2000 or any covered by publications that are systematic reviews or meta-analyses reported after that time. I will focus on human studies, and only cover briefly the huge number of cellular and animal studies. In my judgment the scientific results of greatest importance, consistency and relevance to human health are listed first.

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Is there reliable evidence from epidemiological studies to support the conclusion that low-level RF (below the level at which thermal effects are confirmed) can cause adverse health effects?

There is consistent evidence for harm from low-level RF radiation in studies of individuals using cell phones for prolonged periods of time, which gives a localized exposure to the ipsilateral brain, auditory nerve and parotid gland in the cheek. There have been seven major publications that are either meta-analyses or pooled analyses that evaluate all of the earlier literature, and most find statistically significant relations between elevated exposure to radiofrequency radiation from cell phones and increased risk of brain cancer. I will also discuss several recent individual studies on cell phone exposure and some relevant studies on radio transmission exposure. I will refer frequently to the odds ratio (OR) or risk ratio (RR). These are statistical analysis terms that are used to determine whether or not results are statistically significant. The standard use is to give an OR or RR followed by the 95% confidence interval. Thus, if there is no difference between the "exposed" and "control" populations, the OR or RR will be 1. If there is an elevated risk the OR or RR will be greater than 1.0, whereas if the exposure reduces risk of disease the OR or RR will be less than 1.0. For exposures that increase risk, results are considered to be statistically significant if the 95% CI has a lower bound that is greater than 1, which is to say that there is less than a 5% possibility that the result occurred by chance. The seven major meta-analysis and pooled analysis publications I mentioned are summarized below:

- a. Hardell L, Carlberg M, Soderqvist F, Mild KH. 2008. Meta-analysis of long-term mobile phone use and the association with brain tumours. Internat J Oncology 12: 1097-1103. In ten studies of glioma, cell phone use for more than ten years gave an OR of 1.2 (95%CI=0.8-1.9) (thus this result would not be considered to be significant, since the lower bound is less than 1.0). For ipsilateral cell phone use for more than 10 year the OR = 2.0 (1.2-3.4) (thus this result is statistically significant, since the lower bound is greater than 1.0). There was also a significant association for acoustic neuroma and ipsilateral cell phone use for ten years or more, but no relation for meningioma.
- b. Kundi M. 2009. The controversy about a possible relationship between mobile phone use and cancer. Environ Health Perspect 117: 316-324. Reviewed data from 33 epidemiological studies and concludes that the combined OR = 1.5 (1.2-1.8) for glioma and 1.1 (0.8-1.4) for meningioma.
- c. Myung SK, Ju W, McDonnell DD, Lee YJ, Ksazinet G, Cheng CT, Moskowitz JM. 2009. Mobile phone use and risk of tumors: A meta-analysis. J Clin Oncol 27:5565-5572. Reviewed 465 publications that reported on 12,344 cases of cancer and 25,572 controls. Risk of developing brain cancer was OR = 1.8 (1.04-1.34) for more than ten years use.
- d. Ahlbom A, Feychting M, Green A. Kheifet L, Savitz DA and Swedlow AJ (ICNIRP Standing Committee on Epidemiology). 2009. Epidemiologic evidence on mobile phones and tumor risk: A review. Epidemiology 20: 639-652. Comment that most studies of glioma show small increased or decreased risk among users, although a subset of studies show appreciably elevated risks. They then argue that there are methodological reasons for these positive studies.
- e. Khurana VG, Teo C, Kundi M, Hardell L and Carlberg. 2009. Cell phones and brain tumors: a review including the long-term epidemiological data. Surg Neurol 72: 205-214. Meta-analysis of 11 studies. They conclude that using a cell phone for more than 10 years approximately doubles the risk of being diagnosed with a brain tumor (glioma, OR = 1.9, 1.4-2.4, and acoustic neurona, OR = 1.6, 1.1-2.4) on the ipsilateral side of the head.
- f. Repacholi MH, Lerchl A, Roosli M, Sienkiewica Z, Auvinen A, et al. 2012. Systematic review of wireless phone use and brain cancer and other head tumors. Bioelectromagnetics 33: 187-206. Meta-analysis of

studies shows no relationship between brain cancers and ever use of a mobile phone (for glioma, OR = 1.07, 0.89-1.29, based on eight studies and use for one to five years), but there is sparse data on long-term use. Meta-analysis of oncogenicity, tumor promotion and genotoxicity studies also showed no statistically significant relationship between RF exposure and genotoxic damage to brain cells.

g. Hardell L, Carlberg M, Hansson Mild K. 2012. Use of mobile phones and cordless phones is associated with increased risk for glioma and acoustic neuroma. Pathophysiology doi:10.1016/j.pathophys.2012.11.001. In a review of current evidence they report that a meta-analysis for glioma in the temporal lobe, gave an OR = 1.74 (1.04-2.81). For ipsilateral mobile phone use for 1640 hours or more gave an OR = 2.29 (1.56-3.37). For acoustic neuroma, use for more than 10 years gave an OR = 1.81 (0.73-4.45), and for ipsilateral cumulative us of the same duration the OR = 2.55 (1.50-4.40).

A partial list of recent research studies on cell phone exposure (not reviews) are listed below:

- a. The INTERPHONE Study Group. 2010. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. Int J Epidemiol 39:675-694. While ever vs. never using a cell phone did not increase risk of brain cancer, there was a significant OR= 2.18 (1.43-3.31) for use for ten or more years, OR=1.82 (1.15-2.89) for use for 1640 hours or more and OR=1.31 (0.82-2.11) for more than 270 calls, all for glioma. No significant relations were seen for meningioma. It should be noted that separate INTERPHONE results have been published for Sweden (Lonn et al. 2005. J Epidemiol 161: 526-636) and Germany (Schuz et al. 2006. J Epidemiol 163: 512-520). The German, but not the Swedish study, reported elevated rates of glioma with cell phone use for more than 10 years.
- b. The INTERPHONE Study Group. 2011. Acoustic neuroma risk in relation to mobile telephone use: Results of the INTERPHONE international case-control study. Cancer Epidemiol 35: 453-464. Ever using a cell phone was not associated with elevated risk, nor was use for 10 years or more. For more than 1640 hours of use the OR was 2.79 (1.51-5.16).
- c. Larjavaara S, Schüz J, Swerdlow A, Feychting M, Johansen C, et al. 2011. Location of gliomas in relation to mobile telephone use: A case-case and case-specular analysis. Am J Epidemiol 174: 2-11. Investigated 888

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gliomas from seven European countries (INTERPHONE data) to determine whether the gliomas were located on the side of the head where the cell phone was regularly used. They found an elevated, but not significant, relationship in case-case analysis, but no difference in the case-specular analysis.

- d. Levis AG, Minicuci N, Ricci P, Gennaro V, Gabisa S. 2011. Mobile phones and head tumours. The discrepancies in cause-effect relationships in the epidemiological studies how do they arise? Environ Health 10:59 doi: 10.1186/1476-069X-10-59. When studies that were blinded, free from errors and bias were considered, cell phone use for more than ten years resulted in a near doubling in ipsilateral glioma and acoustic neuroma.
- Aydin D. Feychting M, Schuz J, Tynes T, Andersen TV, et al. 2011. Mobile phone use and brain tumors in children and adolescents: A multicenter case-control study. J Natl Cancer Inst 103: 1264-1276. Studied all children between ages 7-19 with a brain tumor in four European countries. OR for regular mobile phone users was 1.36 (0.92-2.02), and for those using phones at least five years was 1.26 (0.70-2.28). Thus, rates were elevated but not statistically significant and there was no evidence of a dose-response relationship. However, for more than 2.8 years subscription the OR = 2.15 (1.07-4.29), and almost all ORs were elevated when comparing users to non-users. There were highly significant ORs with time since first use, cumulative duration of subscriptions, cumulative duration of call and cumulative number of calls, and these were found on both ipsi- and contralateral sides of the head. This is important, since the evidence for elevated risk only ipsilateral comes from data only on adults, and other evidence indicates greater penetration into the brain of a child. None-theless, the authors conclude that this study provides no support for a relationship between cell phone use and brain cancer in children and adolescents because of the failure to find a dose-response relationship. The conclusions drawn in this study have been questioned by Soderqvist et al. (Environ Health 2011. 10:106) on the basis of the fact that individuals using cordless phones, which generate comparable RF exposure to that from cell phones, was included in the "unexposed" category, and that among the four countries studied ORs for Denmark, Sweden and Switzerland were 1.73, 1.49 and 1.69, respectively, while that for Norway was 0.51. They suggest that this may reflect some methodological difference or bias.
- f. Cardis E, Armstrong BK, Bowman JD, Giles GG, Hours M, et al. 2011. Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries. Occup Environ Med 68:

631-640. ORs for tumours in the most exposed part of the brain in those 1 2 with 10+ years of mobile phone use were 2.80 (1.13-6.94), and were 3 significantly elevated after 7 years of use. The pattern for meningioma was 4 similar but the ORs were lower. 5 6 Frei P, Poulsen AH, Johansen C, Olsen JH, et al. 2011. Use of g. 7 mobile phones and risk of brain tumours: update of Danish cohort study. 8 BMJ doi: 10.1136/bmj.d6387. Used the Danish cancer registry of 3.8 9 million persons. There were 10,729 cases of brain cancer between 1990-10 2007. No increased risk of brain tumors were found among cell phone 11 subscribers as compared to non-subscribers. However, cordless phone 12 subscribers were treated as non-cell phone users in this study. 13 14 Carlberg M, Hardell L. 2012. On the association between glioma. h. 15 wireless phones, heredity and ionizing radiation. Pathophysiology 19: 243-252. Reports on two case-control studies of 1148 glioma cases. They find 16 an OR = 2.9 (1.8-4.7) for ipsilateral use of mobile phones for more than ten 17 18 years. For use of cordless phones they find an OR = 3.8 (1.8-8.1) for 19 ipsilateral use for more than 10 years. ORs were higher for high grade 20 gliomas. Risks were highest among those under age 20. 21 22 There are several reports investigating rates of cancer, particularly leukemia, in 23 persons living near to AM or FM radio transmission towers or cell towers. While 24 most of these studies report elevations in rates of cancer, their assessment of exposure is limited only to residential proximity to the towers, which is not a very 25 26 exact monitor. None-the-less, these studies are significant because they directly 27 monitor rates of human cancer. They also suggest that leukemia is the cancer of 28 greatest concern when the whole body is exposed to radiofrequency radiation, in 29 contrast to more localized cancers with localized exposure. 30 Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, 31 Barca A, Perucci CA. 2002. Adult and childhood leukemia near a high-32 power radio station in Rome, Italy. Am J Epidemiol 155: 1098-1103. The 33 authors show that there is a significant elevation of childhood leukemia

among residents living near to Vatican Radio (Standardized mortality ratio

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= 2.2, 1.0-4.1), and that the risk declines with distance away from the transmitter (p = 0.03).

- b. Eger H, Hagen KU, Lucas B, Vogel P and Voit H. 2004. Einfluss der raumlichen Nahe von Mobilfunksendeanlagen auf die Krebsinzidenz. Umwelt-Medizin-Gellschaft 17: 326-332. A German government-supported study of cancer risk in relation to residence close to cell towers found that rates were significantly higher (OR = 3.38, 95% CI =1.39-8.25; 99% CI = 1.05-10.91) for persons living within 400 m than among those living further away from the towers.
- c. Park SK, Ha M, Im HJ. 2004. Ecological study on residences in the vicinity of AM radio broadcasting towers and cancer death: preliminary observations in Korea. Int Arch Occup Environ Health. 77:387-394. This study found higher mortality areas for all cancers and leukemia in some age groups in the area near the AM towers.
- d. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM, Pack JK. 2007. Radiofrequency radiation exposure from AM radio transmitters and childhood leukemia and brain cancer. Am J Epidemiol 166: 270-279. Leukemia and brain cancer in children in Korea were investigated in relation to residence within 2 km of AM radio transmitters. There was a significant elevation in rates of leukemia (OR = 2.15, 1.00-4.67), but not of brain cancer in relation to peak, but not total radiofrequency exposure for children living within 2 km as compared to more than 20 km from the transmitters.
- e. Merzenich H, Schmiedel S, Bannack S, Bruggemeyer H, Phillipp J, et al. 2008. Childhood leukemia in relation to radio frequency electromagnetic fields in the vicinity of TV and radio broadcast transmitters. Am J Epidemiol 168: 1169-1178. Studied 1,959 cases of leukemia and 5,848 controls in Germany. They did not find any significant relationship between risk of leukemia and living within 2 km of a broadcast transmitter as compared to those living 10-15 km away.
- f. Elliott P, Toledano MB, Bennett J, Beale L, Best N, Briggs DF. 2010. Mobile phone base stations and early childhood cancer: case-control study. BMJ 340: c3077 doi:10.1136/bmj/c3077. No association was found between risk of early childhood cancers and estimates of mother's exposure to mobile phone base stations during pregnancy.
- g. Dode AC, Leao M, Tejo FdeAF, Gomes ACR, Dode DC, Dode MC, Moreira CW, Condessa VA, Albinatti C and Calaffa WT. 2011. Mortality

1 by neoplasia and cellular telephone base stations in the Belo Horizonte 2 municipality, Minas Gerais State, Brazil, Sci Total Environ 409: 3649-3 3665. This study shows higher rates of death from cancer among 4 individuals living close to cell towers than among those living further away. 5 Rates were highest in residences less than 100 m, falling to near 6 background a 1,000 m. 7 8 In summary, the ten major meta-analyses/pooled analyses, the recent cell phone exposure studies, and the radio transmission exposure studies provide convincing 9 10 evidence of adverse health effects in humans associated with low-level RF 11 exposure. Other relevant evidence of human health effects is discussed in 12 Sections 11 and 12 of the *Bioinitiative Report* 2012. 13 Q. Is there evidence about the mechanisms by which low-level RF may adversely 14 affect human physiology? 15 Some, especially those from the physics and engineering community, are skeptical of the ability of radiofrequency radiation to alter human physiological functions 16 17 because of the low energy of the non-ionizing portion of the electromagnetic spectrum. The studies listed below provide evidence that cell phone use and 18 19 applied low-level radiofrequency radiation alter the metabolism of the brain and 20 various clinical measures in humans. They report a variety of effects on humans 21 including dose-dependent changes in cortisol and alpha-amylase, increased brain 22 glucose metabolism, chronic dysregulation of the catacholamine system, and 23 decreases in ACTH, cortisol, thyroid hormones, and prolactin in young females 24 and testosterone in males. 25 Augner C, Hacker GW, Oberfeld G, Florian M, Hitzl W, Hutter J, 26 Pauser G. 2010. Effects of exposure to base station signals on salivary

cortisol, alpha amylase and immunoglobulin A. Biomed Environ Sci 23:199-207. This was a human experimental study with exposure to pulsed wave microwave radiation wherein immune indicators were monitored after five 50-minute sessions. The researchers found dose-dependent changes in cortisol and alpha-amylase.

- b. Volkow ND, Tomasi D, Wange GJ, Vaska P, Fowler JS, Teland F, Alexoff D, Logan J, Wong C. 2011. Effects of cell phone radiofrequency signal exposure on brain glucose metabolism. JAMA 305:808-814. In healthy participants and compared with no exposure, 50-minute cell phone exposure was associated with increased brain glucose metabolism in the region closest to the antenna. This shows direct effects of RF radiation on the brain with cell phone use.
- c. Buchner K, Eger H. 2011. Changes of clinically important neurotransmitters under the influence of modulated RF fields a long-term study under real-life conditions. Umwelt-Medizin-Gesellschaft 24:44-57. There was clear evidence of health-relevant effects, including an increase in adrenaline and noradrenaline, and a subsequent decrease in dopamine in people living near to a new MW-emitting base station. Levels or phenylethylamine decreased and remained decreased, indicating chronic dysregulation of the catacholamine system. Clinically documented increases in sleep problems, headaches, dizziness, concentration problems and allergies followed the onset of new microwave transmissions.
- d. Eskander EF, Estefan SF, Abd-Rabou AA. 2011. How does long term exposure to base stations and mobile phones affect human hormone profiles? Clin Biochem 45:157-161. Measured hormone levels in 82 mobile phone users and 20 controls over a period of 6 years. Report that there were decreases in ACTH, cortisol, thyroid hormones, and prolactin in young females and testosterone in males. There was no change in serum progesterone in females, but in older females prolactin increased with exposure. Exposure from cell phone base stations was associated with significant decreases in ACTH and cortisol.

The following studies report changes in male fertility and reproductive systems associated with cell phone and low-level RF exposure.

a. Wdowiak A, Wdowiak L, Wiktor H. 2007. Evaluation of the effect of using mobile phones on male fertility. Ann Agric Environ Med 14: 169-172. Among Polish males with an infertility problem there was "an increase in the percentage of sperm cells of abnormal morphology associated with duration of exposure to waves emitted by the GSM phone.

It was also confirmed that a decrease in the percentage of sperm cells in vital progressing motility in the semen is correlated with the frequency of using mobile phones."

- b. Agarwal A, Deepinder F, Sharma RK, Ranga G, Li J. 2008. Effect of cell phone usage on semen analysis in men attending infertility clinic: an observational study. Fert Steril 89: 124-128.. "Use of cell phones decreases the semen quality in men by decreasing the sperm count, motility, viability, and normal morphology. The decrease in sperm parameters was dependent on the duration of daily exposure to cell phones and independent of the initial semen quality."
- c. Baste V, Riise T, Moen BE. 2008. Radiofrequency electromagnetic fields: male infertility and sex ratio of offspring. Int J Epidemiol 23:369-377. This is a study of Norwegian Navy personnel chronically exposed to RF fields on the job. The rates of infertility were related to level of exposure in a dose-dependent fashion.
- d. Agarwal A, Desai NR, Makker K, Varghese A, et al. 2009. Effects of radiofrequency electromagnetic waves (RF-EMW) from cellular phones on human ejaculated semen: an *in vitro* pilot study. Fert Stert 92: 1318-1325. "Radiofrequency electromagnetic waves emitted from cell phones may lead to oxidative stress in human semen. We speculate that keeping the cell phone in a trouser pocket in talk mode may negatively affect spermatozoa and impair male fertility.
- e. LaVignera S, Condorelli RA, Vicari E, D'Adata R, Calogero AE. 2012. Effects of the exposure to mobile phones on male reproduction: A review of the literature. J Androl 33: 350-356. Studies in animals and humans show that "RF-EMR decreases sperm count and motility and increases oxidative stress....The results showed that human spermatozoa exposed to RF-EMR have decreased motility, morphometric abnormalities and increased oxidative stress, whereas men using mobile phones have decreased sperm concentration, decreased motility (particularly rapid progressive motility), normal morphology and decreased viability. These abnormalities seem to be directly related to the duration of the mobile phone use."
- f. Avendaño C, Mata A, Sanchez Sarmiento CA, Doncel GF. 2012. Use of laptop computers connected to internet through Wi-Fi decreases human sperm motility and increases sperm DNA fragmentation. Fert Steril 97:39-45. In this study human sperm were exposed to Wi-Fi from a laptop, and were found to show reduced motility after a 4-hour exposure. The

1 results are consistent with other publications (see Agarwal et al., 2008. Fert 2 Steril 89:124-128) that reported that those who use cell phone regularly 3 have reduced sperm count. 4 5 Other evidence of fertility and reproductive effects of low-level RF exposure is 6 discussed in Section 18 of the *Bioinitiative Report 2012*. 7 Q. Is there evidence that some people may become hyper-sensitive to low-level RF 8 and experience related adverse health effects? 9 Electrical hypersensitivity (EHS) is a syndrome of relatively non-specific 10 complaints that are reported to be associated with exposure to electromagnetic 11 fields. The major symptoms are headache, fatigue, tinnitus, disruption of sleep. 12 mental dullness and a general feeling of ill health. Whether or not EHS exists has been widely debated. In spite of widespread reports that up to 10% of the 13 14 population may suffer from EHS, most studies in laboratories with blinded exposures (ie., the subjects do not know whether or not the fields are applied) have 15 16 not demonstrated that persons reporting to be electrosensitive can correctly 17 distinguish when the fields are on. However, there is increasing evidence that 18 EHS does exist and can be a disabling condition for some particularly sensitive 19 persons, although evidence to date is certainly incomplete. 20 There has been only one report of a completely blinded study of an 21 electrosensitive individual that has documented the ability of this individual to report symptoms (primarily headache) in the presence of an electromagnetic field: 22 23 24 McCarty DE, Carrubba S. Chesson AL, Frilor C, Gonzalex-Toledo 25 E. Marino AA. 2011. Electromagnetic hypersensitivity: Evidence for a

1 novel neurological syndrome. Internat J Neurosci 121: 670-676. In a 2 female physician who is electrosensitive, blinded application of 3 electromagnetic fields triggered temporal pain, headache, muscle twitching 4 and skipped heartbeats within 100 seconds of field application. 5 6 There are a number of other reports investigating the prevalence of symptoms in 7 areas near to sources and/or other measures of human response to electromagnetic 8 fields. There are many publications on this subject, and the following are 9 representative of both positive and negative studies: 10 11 Hietanen M, Hamalainen A-M, Husman T. 2002. Hypersensitivity a. 12 symptoms associated with exposure to cellular telephones: No causal link. 13 Bioelectromagnetics 23: 264-270. Studied 20 volunteers who reported themselves to be electrosensitive and exposed them to fields in a blinded 14 15 manner. "None of the test subjects could distinguish real RF exposure from 16 sham exposures." 17 18 Abelin T, Altpeter E, Röösli M. 2005. Sleep disturbances in the b. 19 vicinity of the short-wave broadcast transmitter Schwarzenburg. 20 Somnologie 9:203-209. There is strong evidence of a causal relationship 21 between operation of a short-wave radio transmitter and sleep disturbances 22 in the surrounding population. 23 24 Hutter HP, Moshammer H, Wallner P, Kundi M. 2006. Subjective 25 symptoms, sleeping problems, and cognitive performance in subjects living 26 near mobile phone base stations. Occup Environ Med 63:307-313. There 27 was a significant relation of some symptoms, especially headaches, to 28 measured power density, as well as effects on wellbeing and performance. 29 30 Eliyahu I, Luria R, Hareuveny R, Margaliot M, Neiran N, Shani G. 31 2006. Effects of radiofrequency radiation emitted by cellular telephones on 32 the cognitive functions of humans. Bioelectromagnetics 27:119-266. A 33 total of 36 human subjects were exposed to pulse-modulated microwaves 34 and were tested on four distinct cognitive tasks. Exposure to the left side of 35 the brain slows left-hand response time in three of the four tasks. 36 37 Altpeter ES, Röösli M, Battaglia M, Pfluger D, Minder CE, Abelin 38 T. 2006. Effect of short-wave magnetic fields on sleep quality and 39 melatonin cycle in humans: The Schwarzenburg shut-down study.

Bioelectromagnetics 27:142-150. Sleep quality improved and melatonin excretion increased when the transmitter was shut down.

- f. Preece AW, Georgious AG, Duunn EJ, Farrow SC. 2007. Health response of two communities to military antennae in Cyprus. Occup Environ Med 64:402-408. Compared to residents of a control village, there was a highly significant excess in the reporting of migraine, headache and dizziness in residents living near to military and cell phone antenna systems.
- g. Barth A, Winker R. Ponocny-Seliger E, Mayrhofer W, Ponocny I, Sauter C, Vana N. 2008. A meta-analysis for neurobehavioural effects due to electromagnetic field exposure emitted by GSM mobile phones. Occup Environ Med 65: 342-345. The authors looked at 19 studies of cognitive function in cell phone users, and found in the meta-analysis that there is evidence for a decreased reaction time, altered working memory and increased number of errors in exposed persons.
- h. Landgrebe M, Frick U, Hauser S, Langguth B, et al. 2008. Cognitive and neurobiological alterations in electromagnetic hypersensitive patients: results of a case-control study. Psychol Med 38: 1781-1791. Studies 89 EHS subjects and 107 age and gender matched controls. Found that discrimination ability was significantly reduced in EHS subjects, while intra-cortical facilitation was decreased in younger, but increased in older EHS subjects. They conclude that there are significant cognitive and neurobiological alterations pointing to a higher genuine individual vulnerability in EHS subjects.
- i. Landgrebe M, Frick U, Hauser S, Hajak G, Langguth B. 2009. Association of tinnitus and electromagnetic hypersensitivity: hints for a shared pathophysiology? PLoS One 4: e5026 doi: 10.1371/journal.pone.0005026. Tinnitus occurrence and severity were assessed by questionnaire in 89 EHS and 107 control subjects. Tinnitus was significantly more frequent in the EHS group, but there were no differences in severity or duration. They conclude that tinnitus is associated with subjective EHS.
- j. Furubayashi T, Ushiyama A, Teerao Y, Mizuno Y, et al. 2009. Effects of short-term W-CDMA mobile phone base station exposure on women with or without mobile phone related symptoms. Bioelectromagnetics 30: 100-113. In a double-blind, cross over study of 11 subjects with cell phone-related symptoms and 43 controls, subjected to continuous, intermittent and sham exposure with or without noise, no

 significant effects were found on any psychological, cognitive or autonomic response.

- k. Dahmen N, Ghezel-Ahmadi D, Engel A. 2009. Blood laboratory findings in patients suffering from self-perceived electromagnetic hypersensitivity (EHS). Bioelectromagnetics 30: 299-306. Monitored thyroid hormone, liver enzymes, hemoglobin, hematocrit and c-reactive protein in subjects with and without EHS. "Our results identified laboratory signs of thyroid dysfunction, liver dysfunction and chronic inflammatory processes in small, but remarkable fractions of EHS sufferers."
- l. Eger H, Jahn M. 2010. [Specific health symptoms and cell phone radiation in Selbitz (Bavaria, Germany)- Evidence of a dose-response relationship.] Umwelt-Medizin-Gesellschaft 23: 2. Reports on symptoms of individuals based on residential location and RF measurements of local cell phone radiation levels. "For symptoms as sleep problems, depressions, cerebral symptoms, joint problems, infections, skin problems, cardiovascular problems as well as disorder of the visual and auditory systems and the gastrointestinal tract, a significant dose-response relationship was observed in relation to objectively determined exposure levels".
- m. Robertson JA, Théberge J, Weller J, Drost DJ, Prato FS, Thomas AW. 2010. Low-frequency pulsed electromagnetic field exposure can alter neuro-processing in humans. JR Soc Interface 7:467-473. A functional magnetic resonance imaging study demonstrated how the neuromodulation effect of extremely low-frequency magnetic fields influences the processing of acute thermal pain. The study concludes that magnetoreception may be more common than presently thought. This study was already filed in the present case as Exhibit C-SE-AQLPA-0043, SE-AQLPA-5, Document 10.
- n. Heinrich S, Thomas S, Heumann C, von Kries R and Radon K. 2010. Association between exposure to radiofrequency electromagnetic fields assessed by dosimetry and acute symptoms in children and adolescents: a population based cross-sectional study. Environ Health 9: 75 doi: 10.1186/1476-069X-9-75. The authors studied 1484 children and 1508 adolescents with radiofrequency exposure monitored by a personal dosimeter. Self-reported statistically significant effects found include increased headache (OR 1.50, 1.03-2.19), greater irritation in the evening (OR 1.79, 1.23-2.61) and higher concentrations problems (OR = 1.55, 1.02-2.33) in individuals with greater exposures. However, many others measures did not lead to statistically significant associations.

1 2 Mohler E. Frei P. Braun-Fahrlander C. Frohlich J. et al. 2010. 0. 3 Effects of everyday radiofrequency electromagnetic field exposure on sleep 4 quality: A cross-sectional study. Rad Res 174: 347-356. Studied 1375 5 inhabitants of Basel with a questionnaire and using a prediction model of 6 exposure. "Neither mobile phone use nor cordless phone use was 7 associated with decreased sleep quality." 8 9 Roosli M, Frei P. Mohler E, Hug K. 2010. Systematic review on the p. 10 health effects of exposure to radiofrequency electromagnetic fields from 11 mobile phone base stations. Bull World Health Organ 88: 887-896. 12 Reviewed 17 publications on non-specific symptoms of ill health from RF 13 exposure from mobile phone base stations, and concluded that "At present 14 there is insufficient data to draw firm conclusions about health effects from 15 long-term low-level exposure typically occurring in the everyday 16 environment." 17 18 Papageorgiou CC, Hountala CD, Maganioti AE, Kyprianou MA, 19 Rabavilas AD, Papadimitriou GN, Capsalis CN, 2011. Effects of wi-fi 20 signals on the p300 component of event-related potentials during an auditory Hayling task. J Integr Neurosci 10:189-202. The Hayling 21 22 Sentence Completion test was used to evaluate response initiation and 23 response inhibition. This study concludes that WI-FI exposure may exert 24 gender-related alterations on neural activity. 25 26 Oshima N, Nishida A, Shimodera S, Tochigi M, et al. 2012. The 27 suicidal feelings, self-injury, and mobile phone use after lights out in 28 adolescents. J Pediat Psychol 37: 1023-1030. Studied 17,920 adolescents 29 using a self-report questionnaire. "Logistic regression showed significant 30 associations of the nocturnal mobile phone use with poor mental health. 31 suicidal feelings, and self-injury after controlling for sleep length and other confounders."... "A mechanism of the association might be worsening of 32 33 the quality of sleep." 34 35 In summary, some studies are suggestive of an association, but the reported 36 evidence falls short of proof. In the context of exposure to RF emissions from

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smart meters, there is a substantial body of evidence from the personal accounts of

utility customers who report experiencing EHS symptoms. This evidence should

1 not be disregarded in setting public policy that will determine whether and to what 2 extent people are exposed to these devices. 3 Further discussion of studies of EHS effects can be found in Sections 6 and 4 8 of the Bioinitiative Report 2012. 5 Is there evidence that brain cancer rates have increased in recent decades? Q. 6 Α. If use of cell phones causes brain cancer, then one might expect that overall rates 7 of brain cancer would show an increase, since cell phone use has grown 8 enormously in recent years. However, since use of cell phones is relatively recent 9 and the latency for development of brain cancer following other environmental 10 exposures is long (up to 20-30 years), there might not yet be a clear pattern of 11 increased incidence. The following studies address this issue: 12 13 Central Brain Tumor Registry of the United States (CBTRUS). 14 Supplemental Report: Primary Brain Tumors in the United States, 2004. 15 Hinsdale, IL: Central Brain Tumor Registry of the Unites States 2008. 16 Age-adjusted CNS tumor incidence was 18.2 cases per 100,000 in 2004. 17 but 13.4 cases per 100,000 in 1995. 18 19 b. Lehrer S, Green S, Stock RG. 2010. Association between number of 20 cell phone contracts and brain tumor incidence in nineteen U.S. states. J 21 Neuro-Oncol 101:505-507. "The effect of cell phone subscriptions was 22 significant (P = 0.017), and independent of effect of mean family income (P= 0.894), population (P = 0.003) and age (0.499). The very linear 23 24 relationship between cell phone usage and brain tumor incidence is 25 disturbing and certainly needs further epidemiological evaluation. In the 26 meantime, it would be prudent to limit exposure to all source of electro-27 magnetic radiation." 28 29 De Vocht F, Burstyn I, Cherrie JW. 2011. Time trends (1998-2007) c. 30 in brain cancer incidence rates in relation to mobile phone use in England. 31 Bioelectromagnetics 32:334-339. "There were no time trends in overall 32 incidence of brain cancers for either gender, or any specific age groups.

1 Systematic increases in rates for cancer of the temporal lobe in men... and 2 women... were observed, along with decreases in the rates of cancer of the 3 parietal lobe... and cerebellum..." 4 5 d. Little MP, Curtis RE, Devesa SS, Inskip PD, et al. 2012. Mobile 6 phone use and glioma risk: comparison of epidemiological study results 7 with incidence trends in the United States. BMJ 344: e1147 doi: 8 10.1136/bmj.e1147. "Raised risks of glioma with mobile phone use, as 9 reported by one (Swedish) study forming the basis of the IARC's re-10 evaluation of mobile phone exposure, are not consistent with observed incidence trends in US population data, although US data could be 11 12 consistent with the modest excess risks in the Interphone study." 13 14 Dobes M. Shadbolt B, Khurana VG, Jain S, et al. 2011. A 15 multicenter study of primary brain tumor incidence in Australia (2009-16 2008). Neuro-Oncol 13: 783-790. The authors observed an increased 17 increase in malignant primary brain tumors over the period 2000-2008, but cannot determine whether it was due to improved detection, diagnosis or to 18 19 a true elevated incidence. 20 21 Deltour I, Auviene A, Feychting M, Johansen C, et al. 2012. Mobile 22 phone use and incidence of glioma in the Nordic countries 1979-2008. 23 Epidemiology 23:301-307. "No clear trend change in glioma incidence rates was observed. Several of the risk increases seen in case-control 24 25 studies appear to be incompatible with the observed lack of incidence rate 26 increase in middle-aged men. This suggests longer induction periods than 27 currently investigated, lower risks than reported from some case-control 28 studies, or the absence of any association." 29 30 The Danish Cancer Society recently reported that the number of men 31 who are diagnosed with the most malignant form of brain cancer 32 (glioblastoma) has almost doubled over the past ten years. 33 (http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i 34 +hjernesvulster.htm) 35 36 Further discussion of the relevance of brain cancer rates to the debate about the 37 association between cell phone and RF exposure to cancer is found in Section 11 38 of the Bioinitiative Report, 2012.

In addition to the foregoing evidence of the effects of low-level RF on humans, is 1 Q. 2 there additional evidence from studies of animals and isolated cells? 3 A. Some, but not all studies of isolated cells and intact animals have shown that 4 RF/MW exposures may cause changes in cell membrane function, cell 5 communication, metabolism, activation of proto-oncogenes, and can trigger the 6 production of stress proteins at exposure levels below the above FCC and Health 7 Canada guidelines. Resulting effects in cellular studies include DNA breaks and chromosome aberrations, cell death including death of brain neurons, increased 8 9 free radical production, activation of the endogenous opioid system, cell stress and 10 Additional studies show neurologic, immune, endocrine, premature aging. 11 reproductive and cardiac, adverse health effects from low-dose, chronic exposure 12 to RF/MW radiation in humans. These studies will not be presented here because 13 there are too many and their relevance to human health is uncertain. Please see 14 Bioinitiative Report, 2012 for a comprehensive review of these studies. In 15 summary they do provide additional evidence of biological effects and evidence 16 for possible mechanisms whereby radiofrequency fields may cause adverse health 17 effects including cancer, reproductive and neurobehavioral effects through 18 generation of reactive oxygen species, gene induction and alteration of ion fluxes, 19 but not all positive observations have been fully replicated. 20 Q. Are there any safety standards or guidelines governing RF devices in the 21 United States that are designed to protect people from non-thermal effects of 22 RF exposure?

A. The standards set by the US Federal Communications Commission (FCC) and most international government and non-government organizations are based on the fallacious assumption that there are no adverse human health effects from radiofrequency radiation that does not cause measureable heating. These standards provide no protection whatsoever against non-thermal effects of RF. Some biological effects are known to occur at several hundred thousand times below the FCC public exposure guidelines and the similar guidelines of Health Canada's Safety Code no. 6 (of 6,000,000 $\mu\text{W/m}^2$ or 600 $\mu\text{W/cm}^2$ for the 902-928 MHz bandwidth), as documented in the 2012 *Bioinitiative Report*, Section 24. It is further to be noted that FCC guidelines also apply to 30-minute averaging and Health Canada's Safety Code no. 6 applies to 6-minute averaging. There is no evidence that averaging exposures over time is appropriate for assessing maximum exposure limits to low-level RF.

Furthermore, these limits are based on the incorrect biological assumption that body temperatures must increase at least 1°C to lead to potential biological impacts and the impacts of absorbing RF within the band of the electromagnetic spectrum that smart meters use would only be limited to behavioral disruption. These limits do not take into account the scientific research that show tissue heating may result in many adverse health effects other than "behavioral disruption". These limits also do not take into account the accepted biological fact that every enzyme system in the body is exquisitely sensitive to temperature and may increase activity by even a fraction of a degree increase in temperature. What

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is defined as "non-thermal" effect is therefore partly a function of our ability to measure the temperature increase. See Bioinitiative Report, Section 24 for further discussion. FCC public RF/MW radiation exposure guidelines (and the similar Health Canada Safety Code no. 6 guidelines) are based on the height, weight and stature of a 6-foot tall man, not children or adults of smaller stature. The guidelines do not take into account the unique susceptibility of growing children to RF/MW radiation exposures. Since children are growing, their rate of cellular activity and division is more rapid, and they are at a greater risk for DNA damage and subsequent cancers. Growth and development of the central nervous system is still occurring well into the teenage years, such that the neurological impairments predictable by the extant science may have great impact upon development. cognition, learning, and behavior. Q. Have you reviewed the joint testimony of William H. Bailey, Ph.D. and Yakov Shkolnikov, Ph.D., dated September 19, 2012? A. Yes. In their testimony, Dr. Bailey and Dr. Shkolnikov cite a report by the Q. ICNIRP Committee, which concluded that "the trend in the accumulated evidence is increasingly against the hypothesis that mobile phone use causes brain tumors." Do you agree with that conclusion? Α. I strongly disagree. The weight of evidence indicates that mobile phone use is associated with elevated risk of brain cancer which becomes apparent after ten or

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more years of intensive use and occurs primarily on the side of the head where the user holds his/her phone the majority of the time. There is emerging evidence that younger people are at greater risk than older individuals. The great majority of the meta-analyses that have been published on the subject demonstrate a statistically significant elevation in rates of brain cancer with long-term cell phone use. This statement by Bailey and Shkolnikov is simply not true.

It is necessary to comment on the ICNIRP report, as well as on the UK Advisory Group on Non-Ionising Radiation (AGNIR) report, published in April, 2012, which is also cited by Bailey and Shkolnikov. It should be noted that there is considerable overlap in the membership of these two groups. Both ignore or attempt to discredit the information presented above. The AGNIR report fails to even mention the IARC classification of radiofrequency fields as possible human carcinogens. Neither is a fair and balanced review of the scientific evidence concerning the human health effects of radiofrequency fields. A much more convincing review of the evidence is found in the Ramazzini Institute European Journal of Oncology Library, Volume 5, entitled "Non-thermal effects and mechanisms of interaction between electromagnetic fields and living matter," published in 2010, and in the *Bioiniative Report*, 2012. The primary reason that I and the other authors prepared the *Bioinitiative Report* was and is to counter the prejudicial and false conclusions of these reports, and to do so by presenting a comprehensive review of scientific evidence.

Q. Do you agree with their testimony that the authors of the *Bioinitiative Report*used flawed methods and failed to follow "the standard, scientific methods for
developing exposure limits."

A.

I strongly disagree with this statement. It should be noted that the *Bioinitiative Report* does not recommend exposure limits *per se*, but rather identifies exposures levels which are associated with biological effects, some of which are adverse effects on human health. The public health chapter, of which I am a co-author, identifies a "no observed effect level" (NOEL), based on the scientific evidence from peer-reviewed scientific studies, then applies safety factors for sensitive populations (the fetus, children, the aged, etc.) as is standard practice in chemical risk assessment. This chapter presents clear documentation of why more stringent limits on exposure are necessary to protect human health.

The *BioInitiative Report* is aimed at *restoring* the balance, by providing a more comprehensive review of the evidence. The *Bioinitiative Report* mentions many negative reports, discusses the weight of evidence, and looks for inconsistencies. For example, Prof. Henry Lai of the University of Washington in the 2012 *Bioinitiative Report* presents summaries of 86 scientific studies on genotoxic effects of radiofrequency radiation published since 2007, and finds that 63% of these found statistically significant positive effects, while of 155 new studies on neurological effects, 98 found effects. The *Bioinitiative Report*, unlike either the ICNIRP or AGNIR reports, reviews of the scientific research available, both those showing and not showing biological effects and human disease, and

draws conclusions based on the weight of the evidence that standard setting organizations were failing to properly take into account.

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Q. Dr. Bailey and Dr. Shkolnikov testified that: "The weight of the evidence does not support the idea that significant biological or adverse health effects can occur" from RF exposure. Do you agree with this conclusion?

This statement is almost incomprehensible given the strength of the evidence demonstrating consistent and serious adverse health effects in both animal and human studies. The studies of greatest importance are those which demonstrated elevations in cancers, especially leukemia and brain cancer, in association with exposure to radiofrequency EMFs. There is evidence that exposure to cell phone frequencies increased uptake of glucose in the brain, which indicates that RF radiation alters fundamental process within the nervous system. The thousands of studies in cellular and animal systems provide additional evidence that radiofrequency fields alter a host of biochemical, physiological and behavioral factors. While certainly not every study reports positive and statistically significant results, the majority do as clearly documented in the 2012 Biointiative Report. No objective person could possibly make a statement such as this if they are at all familiar with the literature published in high-quality, peer-reviewed scientific journals, and if they are coming to the question with an open mind without a major conflict of interest.

Standards setting organizations aimed at regulating RF exposure have for a long time been dominated by physicists and engineers, often with close ties with the industry, with little input from biological and medical science. In spite of evidence to the contrary, many such people have as a statement of faith that RF fields that do not cause measureable tissue heating cannot have biologic effects. This point of view is incompatible with the science. Standards setting organizations also often explicitly take into account the economic impacts of the standards when faced with scientific uncertainty. Both because of their training and because of their ties with the industry, members of most of these organizations have been reluctant to take the above biological findings into account when proposing exposure limits.

These organizations have generally refused to accept epidemiological and laboratory research findings linking RF electromagnetic fields exposure with various non-thermal biological effects, as being inconclusive and requiring further research. The difficulty stems from the fact that, although links have been demonstrated repeatedly between RF electromagnetic fields exposure and non-thermal biological effects in humans, there is a lack of a comprehensive biological theory explaining why these effects take place, and therefore causality cannot, at the present time, be demonstrated with certainty. Animals do not always respond to RF electromagnetic fields as do humans. Also, in some cases, experimental results in cellular studies have not been replicated in other laboratories; in some cases attempts to duplicate results showed negative results or variations in the

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results. These discrepancies are, however, normal in the research process and may result from slight, but significant differences in procedures; they indicate that biological systems are complex and that different variables need to be isolated in order to fully understand these systems. Research is still needed in order to determine to what extent non-thermal biological effects may vary with frequency, with modulation and depend on the pulsed (instead of continuous) character of RF emissions. There may also be variance between the levels of reaction of different subjects for reasons that still remain to be explained. This is what the research process is about. In biology and medicine there is nothing that is 100% proven: our understanding of various illnesses, cancer and Alzheimer's, for example, is still largely incomplete. We rely on statistical significance and weight of evidence and, therefore, on judgment, when drawing conclusions about health effects. In your opinion, could a careful scientist familiar with the body of knowledge on the subject reliably conclude that there are no risks of adverse health effects from the exposure to RF in the 2.4 GHz range? On the basis of the vast body of scientific literature, many public health experts, myself included, are of the opinion that exposure to RF/MW radiation and EMFs, including in the range of 2.4GHz, poses a potential of serious threat to public health. The degree of risk will vary with both the intensity and duration of exposure. It is likely society will face markedly increased incidence of neurotoxic effects, neurodegenerative diseases, cancers and genotoxicity in the future,

1 resulting from the extreme and mostly involuntary exposure to RF/MW radiation 2 and EMFs. 3 Q. Are you familiar with smart meter technology? 4 A. I am familiar with smart meter technology. 5 Q. In your opinion, could a careful scientist familiar with the body of knowledge 6 on the subject reliably conclude that there are no risks of adverse health 7 effects from exposure to RF from smart meters emitting RF radiation in the 2.4 GHz range with peak power densities of approximately 0.44 mW/cm²? 8 9 A. There are two types of smart meter technology. Wired smart meters pose no risk 10 of exposure to RF radiation. Wireless smart meters, on the other hand, pose a 11 substantial risk of RF exposure which is dependent on the frequency of pulsed RF. 12 the intensity of the pulsed RD and the individual's distance from the meter. While 13 there have not been human health studies done to date of the effects of exposure to 14 smart meter RF, because the technology is too new and the latency for adverse 15 effects for diseases such as cancer is long, the evidence from the cell phone studies 16 demonstrates convincingly that wireless smart meters pose a risk to human health. 17 Smart meters send pulsed RF radiation at intermittent periods of time. 18 While the frequency of these pulses may vary with different smart meters, some 19 have been reported to send pulses over 30 times a minute at peak power density reading of over 67mW/m^2 (0.0067mW/cm²) (Maisch. 2012. Smart meter health 20 21 concerns: Just a nocebo effect or an emerging public health nightmare? ACNEM 22 Journal 31: 15-19), and this exposure has been associated with self-reported

experimental studies that provide some of the evidence of low intensity exposure effects from radiofrequency radiation at low-intensity exposures. Because the meters operate intermittently 24/7, an individual in the vicinity of the meter will be continuously exposed to RF.

It is correct that the CMP smart meters comply with the FCC standard of 1 mW/cm². The problem is that the FCC standard is based on the assumption that there are no effects of RF radiation other than tissue heating, which is simply not the case.

For most smart meter use, the cumulative average RF exposure is not great, but the reported health effects are large. This raises the important question as to whether the exposure of greatest concern is the cumulative average, or rather the peak power levels in the pulses. This issue is discussed in Chapter 24 of the 2012 *Biointiative Report*, which presents some evidence that it is the peak power that is important. However, the total exposure will only increase in the future as RF devices are being placed in every appliance in the home, and will use RF to communicate to the smart meter which will communicate with the utility. This will make the home, especially the kitchen, a source of highly elevated RF exposure whenever an appliance is used.

Further investigation of the human health effects of smart meter exposures is essential. In the meantime it is extremely unwise to implement the smart grid with wireless smart meters until we understand fully the potential for harm to human health.

Dated this 18th day of January, 2013.

Marid Slar fon Yen David O. Carpenter, M.D.

STATE OF NEW YORK RENSSELAER, ss:

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January 22, 2013

Personally appeared the above-named David O. Carpenter, M.D., and stated under oath that the foregoing Affidavit made by him is true and based upon his own personal knowledge, information or belief, and so far as upon information and belief, he believes the information to be true. Before me,

Notary Public/Attorney-at-Law Dorcen A Van Vorst

Name Typed or Printed My Commission Expires:

DOREEN A. VanVORST
Notary Public, State of New York
Qualified in Reneselaer County
Reg. No. 01VA5083834
My Commission Expires Aug. 25, 20/3

CURRICULUM VITAE

Name:

David O. Carpenter

Home Address:

2749 Old State Road

Schenectady, New York 12303

Positions Held:

Director, Institute for Health and the Environment

University at Albany

Professor, Environmental Health Sciences School of Public Health, University at Albany 5 University Place, A217, Rensselaer, NY 12144

Honorary Professor

Queensland Children's Medical Research Institute

University of Queensland Brisbane, Australia

Education:

1959 1964 B.A., Harvard College, Cambridge, MA M.D., Harvard Medical School, Boston, MA

Positions Held:

York

9/61-6/62	Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg
7/64-6/65	Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman
7/65-2/73	Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.
2/73-3/80	Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD
3/80-9/85	Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
9/85-1/98	Dean, School of Public Health, University at Albany
9/85-Pres.	Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.
9/85-7/98	Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY
1/98-1/05	Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY
2001-Pres.	Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.
2005-Pres.	Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New

Editor-in-Chief:

Cellular and Molecular Neurobiology, 1981 – 1987

Editor-in Chief:

Reviews on Environmental Health 2012-present

Editor-in-Chief: Editorial Advisor:

<u>Journal of Local and Global Health Sciences</u> 2012-present Cellular and Molecular Neurobiology, 1987 - Present

Editorial Boards:

<u>Journal of Public Health Management and Practice</u>, 1995 - 2002 International Journal of Occupational Medicine & Environmental Health

1996 - Present

Journal of Alzheimer's Disease - Associate Editor, 2007-2009

Reviews in Environmental Health; 2008-2012

International Archives of Occupational and Environmental Health; 2009-present.

Journal of Environmental and Public Health, 2009-present.

Environmental Health Perspectives, 2010-present

Global Health Perspective, 2012-present

National and International Committees:

1978, 1981 1979-1985 1974-1981	Physiology Study Section (Ad hoc member) NIH International Fellowship Study Section Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
1981-1989 1985-1986	Member, USA National Committee for the International Brain Research Organization Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
1986-1987	Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
1987-1989	Member, Science Advisory Council of the American Paralysis Association
1987-1990	Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
1985-1993	Committee #79, National Council on Radiation Protection and Measurements
1986-1997	Member, Legislative and Education Committees, Association of Schools of Public Health
1989-1994	Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
1994, 1995	Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
1994-1997	Member, Legislative Committee of the Association of Schools of Public Health
1997	Member, Executive Committee of the Association of Schools of Public Health
1997-2000	National Advisory Environmental Health Sciences Council of the National Institutes of Health
1998-Pres.	Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
2000-Pres.	Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
2001-2008	United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
2002-2003	Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
2003-2008	Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
2003-Pres.	Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
2004-Pres. 2007-2011	Member, Blue Ocean Institute Curriculum Advisory Board. Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board,
2007-2011	International Joint Commission.

State and Local Committees:

1980-1987 1985-1989	Executive Secretary, New York State Power Lines Project Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
1986-1989	Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
1991-1992	Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
1991-1992	Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
1991-1992	Member, State Task Force for the Reform of Middle Level Education in NY State
1992-1993	Member, State Needs Task Force on Health Care and Education
1987-1998	Delegate-at-Large, New York State Public Health Association
1991-1995	Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
1994	Chair, Council of Deans, University at Albany, SUNY
1997-2008.	Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
2000-Pres.	Member, Board of Directors, Healthy Schools Network, Inc.
2000-2003	Member, Medical Advisory Board, Hepatitis C Coalition, New York
2000-2004	Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
2001-2008	Member, Board of Directors, Environmental Advocates of New York
2004-2007	Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
2005-Pres.	Member, Schooling Chefs Curriculum Advisory Board
2005-Pres.	Member, Advisory Board, Healthy Child Healthy World
2005-2008	Member, Board of Directors, Citizens Environmental Coalition
2006-2009	Member, Board of Directors, Marine Environmental Research Institute
2007-2009	Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

1959	B.A. awarded <u>magna cum laude</u> . Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, <u>Ambystoma tigrinum</u> " (Thesis advisor, Professor George Wald)
	Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded <u>cum laude</u> for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu
1987	University, Fukuoka, Japan, for a period of three months each
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies

	in environmental health.
2001	Awarded 2001 Academic Laureate from the University at Albany Foundation.
2010	Awarded the Albion O. Bernstein, M.D. Award in recognition of an outstanding contribution to public health and the prevention of disease though lifelong research of environmental health hazards and for limitless devotion to medical education by the Medical Society of the State of New York.
2011	Awarded the Rodney Wylie Eminent Visiting Fellowship 2011 at the University of Queensland, Brisbane, Australia for a period of four weeks.
Federal Grai	nts Held: (Principal Investigator Only)
1980-1983	United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs", \$76,847 total direct costs.
1982-1988	National Institute of Health, "Mechanisms of Desensitization at Central Synapses", \$464,786 total direct costs.
1984-1986	Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs@, \$330,504 total direct costs.
1986-1996	National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct costs.
1989-1993	National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total direct costs
1990-1995	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O. Carpenter, P.I. \$5,783,419 total direct costs.
1995-2001	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. ACentral/Eastern European Environ/Occup Training Program@, D.O. Carpenter, P.I. \$657,520 total costs.
1995-2001	National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total direct costs.
1998-1999	Environmental Protection Agency, Alndoor Air Risk at Akwesasne - Pilot Project@, D.O. Carpenter, P.I. \$9,996 total costs.
2000-2002	Association Liaison Office for University Cooperation in Development, ACooperative Program in Environmental Health between the Institute of Public Health at Makerere University, Kampala, Uganda and the School of Public Health, University at Albany, USA@, D.O. Carpenter, P.I. \$96,432 total costs.
2001-2007	Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. AMultidisciplinary Environmental Health Training@, D.O. Carpenter, P.I. \$850,000 total costs.
2006-2011	Pakistan-US Science and Technology Cooperative Program (US National Academy of Sciences). "Association of particulate matter with daily morbidity in an urban population," D.O. Carpenter, P.I., \$391,104 total costs.
2009-2013	Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2:
	Environmental contaminants and reproductive health of Akwesasne Mohawk women.

\$387,825 for year 1. D.O. Carpenter, Co-Pl.

- 2010-2013 Department of the Army, "Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, "Drinking Water Supply, Sanitation, and Hygiene Promotion: Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda". D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1RO1ES019620), "Protecting the health of future generations: Assessing and preventing exposures." PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2,354,871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson's Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, <u>The Public Radio Health Show</u> (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001.

Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

- 1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
- 2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
- 3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. <u>Acta Physiol. Scand.</u>, 59:126-142.
- 4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
- 5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. <u>Acta Physiol. Scand.</u>, 63:103-110, 1965.
- 6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibitibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.

- 7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. <u>J. Neurophysiol.</u>, 28:560-580, 1965.
- 8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.
- 9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. <u>J. Neurophysiol.</u>, 28:958-965, 1965.
- 10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. <u>Arch. Ital. Biol.</u>, 104:73-85, 1966.
- 11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J. Neurophysiol., 29:353-368, 1966.
- 12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in <u>Aplysia</u> neurons. <u>J. Gen. Physiol.</u>, 50:1469-1484, 1967.
- 13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. Pharmacol., 6A:351-364, 1968.
- 14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na⁺ pump to membrane potential in Aplysia neurons. J. Gen. Physiol., 52:1-21, 1968.
- 15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.
- 16. Carpenter, D.O. Membrane potential produced directly by the Na⁺ pump in <u>Aplysia</u> neurons. <u>Comp. Biochem. Physiol.</u>, 35:371-385, 1970.
- 17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of <u>Aplysia</u> neurons upon Na⁺ and Ca⁺⁺. <u>J. Cell. Physiol.</u>, 75:121-127, 1970.
- 18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepin-ephrine in the presence of tetrodotoxin. <u>J. Pharmacol. Exp. Therap.</u>, 73:416-421, 1970.
- 19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.
- 20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of <u>Aplysia</u> neurons and squid axon as determined by a new technique. Intl. J. Neurosci., 2:35-48, 1971.
- 21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in <u>Aplysia</u> nervous and non-nervous tissues. <u>Int. J. Neurosci.</u>, 2:49-56, 1971.
- 22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of <u>Aplysia</u> neuron somata. <u>Science</u>, 176:1329-1331, 1972.
- 23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. Science, 179:1336-1338, 1973.
- 24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. J. Physiol. (Lond.), 229:471-493, 1973.
- 25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. <u>Aplysia</u> acetylcholine receptors: Blockage by and binding of α-bungarotoxin. <u>Brain Res.</u>, 72:225-240, 1974.
- 26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. Brain Res., 73:156-160, 1974.
- 27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in <u>Aplysia</u> suggests neurotransmitter function. <u>Science</u>, 185:364-365, 1974.

- 28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of <u>Aplysia</u> neurons. <u>J. Cell. Physiol.</u>, 84:463-472, 1974.
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- 35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents in vitro. <u>Pflugers Arch.</u>, 359:349-356, 1975.
- 36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on <u>Aplysia</u> neurons. Nature, 258:751-754, 1975.
- 37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on <u>Aplysia</u> neurons. <u>Science</u>, 192:806-809, 1976.
- 38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. Biophys. J., 16:991-1001, 1976.
- 39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α-bungarotoxin with <u>Aplysia</u> acetylcholine receptors. <u>Mol. Pharmacol.</u>, 12:999-1006, 1976.
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- 55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthamol., 89:641-646, 1980.
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- 73. Carpenter, D.O., Briggs, D.B. and Strominger, N. Responses of neurons of canine area postrema to neurotransmitters and peptides. <u>Cell. Molec. Neurobiol.</u>, 3:113-126, 1983.
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Power Density (Microwatts/centimeter2 - uW/cm2)	ter2 - uW/cm2)	Reference
As low as (10 ⁻¹³) or 100 femtowatts/cm2	Super-low intensity RFR effects at MW reasonant frequencies resulted in changes in genes; problems with chromatin conformation (DNA)	Belyaev, 1997
5 picowatts/cm2 (10-	Changed growth rates in yeast cells	Grundler, 1992
0.1 nanowatt/cm2 (10- ¹⁰) or 100 picowatts/cm2	Super-low intensity RFR effects at MW reasonant frequencies resulted in changes in genes; problems with chromatin condensation (DNA) intensities comparable to base stations	Belyaev, 1997
0.00034 uW/cm2	Chronic exposure to mobile phone pulsed RF significantly reduced sperm count,	Behari, 2006
0.0005 uW/cm2	RFR decreased cell proliferation at 960 MHz GSM 217 Hz for 30-min exposure	Velizarov, 1999
0.0006 - 0.0128 uW/cm2	Fatigue, depressive tendency, sleeping disorders, concentration difficulties, cardio- vascular problems reported with exposure to GSM 900/1800 MHz cell phone signal at base station level exposures.	Oberfeld, 2004
0.0009 uW/cm2	RFR induced 10%-40% increase in DNA synthesis in glioma cells (brain)	Stagg, 1997
0.003 - 0.02 uW/cm2	In children and adolescents (8-17 yrs) short-term exposure caused headache, irritation, concentration difficulties in school.	Heinrich, 2010
0.003 to 0.05 uW/cm2	In children and adolescents (8-17 yrs) short-term exposure caused conduct problems in school (behavioral problems)	Thomas, 2010
0.005 uW/cm2	In adults $(30-60 \text{ yrs})$ chronic exposure caused sleep disturbances, (but not significantly increased across the entire population)	Mohler, 2010
0.005 - 0.04 uW/cm2	Adults exposed to short-term cell phone radiation reported headaches, concentration difficulties (differences not significant, but elevated)	Thomas, 2008
0.006 - 0.01 uW/cm2	Chronic exposure to base station RF (whole-body) in humans showed increased stress hormones; dopamine levels substantially decreased; higher levels of adrenaline and nor-adrenaline; dose-response seen; produced chronic physiological stress in cells even after 1.5 years.	Buchner, 2012
0.01 - 0.11 uW/cm2	RFR from cell towers caused fatigue, headaches, sleeping problems	Navarro, 2003

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

CARPENTER EXHIBIT B

(Microwatts/centimeter2 - uW/cm2)	er2 - uW/cm2)	Reference
0.01 - 0.05 uW/cm2	Adults (18-91 yrs) with short-term exposure to GSM cell phone radiation reported headache, neurological problems, sleep and concentration problems.	Hutter, 2006
0.005 - 0.04 uW/cm2	Adults exposed to short-term cell phone radiation reported headaches, concentration difficulties (differences not significant, but elevated)	Thomas, 2008
0.015 - 0.21 uW/cm2	Adults exposed to short-term GSM 900 radiation reported changes in mental state (e.g., calmness) but limitations of study on language descriptors prevented refined word choices (stupified, zoned-out)	Augner, 2009
0.05 - 0.1 uW/cm2	RFR linked to adverse neurological, cardio symptoms and cancer risk	Khurana, 2010
0.05 - 0.1 uW/cm2	RFR related to headache, concentration and sleeping problems, fatigue	Kundi, 2009
0.07 - 0.1 uW/cm2	Sperm head abnormalities in mice exposed for 6-months to base station level RF/MW. Sperm head abnormalities occurred in 39% to 46% exposed mice (only 2% in controls) abnormalities was also found to be dose dependent. The implications of the pin-head and banana-shaped sperm head. The occurrence of sperm head observed increase occurrence of sperm head abnormalities on the reproductive health of humans living in close proximity to GSM base stations were discussed."	Otitoloju, 2010
0.38 uW/cm2	RFR affected calcium metabolism in heart cells	Schwartz, 1990
0.8 - 10 uW/cm2	RFR caused emotional behavior changes, free-radical damage by super-weak MWs	Akoev, 2002
0.13 uW/cm2	RFR from 3G cell towers decreased cognition, well-being	Zwamborn, 2003
0.16 uW/cm2	Motor function, memory and attention of school children affected (Latvia)	Kolodynski, 1996
0.168 - 1.053 uW/cm2	Irreversible infertility in mice after 5 generations of exposure to RFR from an 'antenna park'	Magras & Zenos, 1997
0.2 - 8 uW/cm2	RFR caused a two-fold increase in leukemia in children	Hocking, 1996
0.2 - 8 uW/cm2	RFR decreased survival in children with leukemia	Hocking, 2000
0.21 - 1.28 uW/cm2	Adolescents and adults exposed only 45 min to UMTS cell phone radiation reported increases In headaches.	Riddervold, 2008

Cardiac, heart muscle, blood-pressure, vascular effects	Disrupted calcium metabolism
Cancer (other than brain), cell proliferation	Oxidative damage/ROS/DNA damage/DNA repair failure
Sleep, neuron firing rate, EEG, memory, learning, behavior	Reproduction/fertility effeccts
Brain tumors and blood-brain barrier	Stress proteins, HSP, disrupted immune function

CARPENTER EXHIBIT B

Power Density (Microwatts/centimeter2 - uW/cm2)	meter2 - uW/cm2)	Reference
0.5 uW/cm2	Significant degeneration of seminiferous epithelium in mice at 2.45 GHz, 30-40 min.	Saunders, 1981
0.5 - 1.0 uW/cm2	Wi-FI level laptop exposure for 4-hr resulted in decrease in sperm viability, DNA fragmentation with sperm samples placed in petri dishes under a laptop connected via WI-FI to the internet.	Avendano, 2012
1.0 uW/cm2	RFR induced pathological leakage of the blood-brain barrier	Persson, 1997
1.0 uW/cm2	RFR caused significant effect on immune function in mice	Fesenko, 1999
1.0 uW/cm2	RFR affected function of the immune system	Novoselova, 1999
1.0 uW/cm2	Short-term (50 min) exposure in electrosensitive patients, caused loss of well-being after GSM and especially UMTS cell phone radiation exposure	Eltiti, 2007
1.3 - 5.7 uW/cm2	RFR associated with a doubling of leukemia in adults	Dolk, 1997
1.25 uW/cm2	RFR exposure affected kidney development in rats (in-utero exposure)	Pyrpasopoulou, 2004
1.5 uW/cm2	RFR reduced memory function in rats	Nittby, 2007
2 uW/cm2	RFR induced double-strand DNA damage in rat brain cells	Kesari, 2008
2.5 uW/cm2	RFR affected calcium concentrations in heart muscle cells	Wolke, 1996
2 - 4 uW/cm2	Altered cell membranes; acetycholine-induced ion channel disruption	D'Inzeo, 1988
4 uW/cm2	RFR caused changes in hippocampus (brain memory and learning)	Tattersall, 2001
4 - 15 uW/cm2	Memory impairment, slowed motor skills and retarded learning in children	Chiang, 1989
5 uW/cm2	RFR caused drop in NK lymphocytes (immune function decreased)	Boscolo, 2001
5.25 uW/cm2	20 minutes of RFR at cell tower frequencies induced cell stress response	Kwee, 2001
5 - 10 uW/cm2	RFR caused impaired nervous system activity	Dumansky, 1974
6 uW/cm2	RFR induced DNA damage in cells	Phillips, 1998

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

Power Density (Microwatts/centimeter2 - uW/cm2)	neter2 - uW/cm2)	Reference
8.75 uW/cm2	RFR at 900 MHz for 2-12 hours caused DNA breaks in leukemia cells	Marinelli, 2004
10 uW/cm2	Changes in behavior (avoidance) after 0.5 hour exposure to pulsed RFR	Navakatikian, 1994
10 - 100 uW/cm2	Increased risk in radar operators of cancer; very short latency period; dose response to exposure level of RFR reported.	Richter, 2000
12.5 uW/cm2	RFR caused calcium efflux in cells - can affect many critical cell functions	Dutta, 1989
13.5 uW/cm2	RFR affected human lymphocytes - induced stress response in cells	Sarimov, 2004
14.75 uW/cm2	RFR increased biomarker for cell division in glioma brain tumor cells	Stagg, 1997
20 uW/cm2	Increase in serum cortisol (a stress hormone)	Mann, 1998
28.2 uW/cm2	RFR increased free radical production in rat cells	Yurekli, 2006
37.5 uW/cm2	Immune system effects - elevation of PFC count (antibody producing cells	Veyret, 1991
45 uW/cm2	Pulsed RFR affected serum testosterone levels in mice	Forgacs, 2006
50 uW/cm2	Cell phone RFR caused a pathological leakage of the blood-brain barrier in 1 hour	Salford, 2003
50 uW/cm2	An 18% reduction in REM sleep (important to memory and learning functions)	Mann, 1996
60 uW/cm2	RFR caused structural changes in cells of mouse embryos	Somozy, 1991
60 uW/cm2	Pulsed RFR affected immune function in white blood cells	Stankiewicz, 2006
60 uW/cm2	Cortex of the brain was activated by 15 minutes of 902 MHz cell phone	Lebedeva, 2000
65 uW/cm2	RFR affected genes related to cancer	Ivaschuk, 1999
92.5 uW/cm2	RFR caused genetic changes in human white blood cells	Belyaev, 2005
100 uW/cm2	Changes in immune function	Elekes, 1996
100 uW/cm2	A 24.3% drop in testosterone after 6 hours of CW RFR exposure	Navakatikian, 1994

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

Power Density (Microwatts/centimeter2 - uW/cm2)	eter2 - uW/cm2)	Reference
120 uW/cm2	A pathological leakage in the blood-brain barrier with 915 MHz cell RF	Salford, 1994
500 uW/cm2	Intestinal epithelial cells exposed to 2.45 GHz pulsed at 16 Hz showed changes in intercellular calcium.	Somozy, 1993
500 uW/cm2	A 24.6% drop in testosterone and 23.2% drop in insulin after 12 hrs of pulsed RFR exposure.	Navakatikian, 1994
STANDARDS		.50
530 - 600 uW/cm2	Limit for uncontrolled public exposure to 800-900 MHz	ANSI/IEEE and FCC
1000 uW/cm2	PCS STANDARD for public exposure (as of September 1,1997)	FCC, 1996
5000 uW/cm2	PCS STANDARD for occupational exposure (as of September 1, 1997)	FCC, 1996
BACKGROUND LEVELS	LS	
0.003 uW/cm2	Background RF levels in US cities and suburbs in the 1990s	Mantiply, 1997
0.05 uW/cm2	Median ambient power density in cities in Sweden (30-2000 MHz)	Hamnierius, 2000
0.1 - 10 uW/cm2	Ambient power density within 100-200' of cell site in US (data from 2000)	Sage, 2000

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

	750 MHz continuous wave (CW) RFR exposure caused increase in heat shock protein (stress proteins).	0 001 W/K
Lass, 2002	MW modulated at 7 Hz produces more errors in short-term memory functioin on complex tasks (can affect cognitive processes such as attention and memory)	0.0095 W/Kg
Marinelli, 2004	900 MHz cell phone signal induces DNA breaks and early activation of p53 gene; short exposure of 2-12 hours leads cells to acquire greater survival chance - linked to tumor agressiveness.	0.0035 W/Kg
Navakatikian, 1994	Changes in active avoidance conditioned behavioral effect is seen after one-half hour of pulsed radiofrequency radiation	0.0027 W/Kg
Phillips, 1998	Digital cell phone RFR at very low intensities causes DNA damage in human cells; both DNA damage and impairment of DNA is reported	0.0024 - 0.024 W/Kg
Kwee, 2001	Heat shock protein HSP 70 is activated by very low intensity microwave exposure in human epithelial amnion cells	0.0021 W/Kg
Tattersall, 2001	Very low power 700 MHz CW affects excitability of hippocampus tissue, consistent with reported behavioral changes.	0.0016 - 0.0044 W/Kg
Stagg, 1997	Changes in brain glial cells with TDMA 836.55 MHz frequency	0.0009 W/Kg
Aldad, 2012	Neurobehavioral disorders in offspring of pregnant mice exposed in utero to cell phones - dose-response impaired glutamatergic synaptic transmission onto layer V pyramidal neurons of the prefrontal cortex. Hyperactivity and impaired memory function in offspring. Altered brain development.	0.0003 - 0.06 W/Kg
Kwee, 1997	Changes in cell cycle; cell proliferation (960 MHz GSM mobile phone)	0.000021 - 0.0021 W/Kg
Schwartz, 1990	Calcium ion movement in isolated frog heart tissue is increased 18% (P<.01) and by 21% (P<.05) by weak RF field modulated at 16 Hz $$	0.00015 - 0.003 W/Kg
TNO Physics and	Well-being and cognitive function affected in humans exposed to GSM-UMTS cell phone frequencies; RF levels similar near cell sites	0.000064 - 0.000078 W/Kg
Reference		SAR (Watts/Kilogram)

Cardiac, heart muscle, blood-pressure, vascular effects	Disrupted calcium metabolism
Cancer (other than brain), cell proliferation	Oxidative damage/ROS/DNA damage/DNA repair failure
Sleep, neuron firing rate, EEG, memory, learning, behavior	Reproduction/fertility effeccts
Brain tumors and blood-brain barrier	Stress proteins, HSP, disrupted immune function

Reported Biological Effects from Radiofrequency Radiation at Low-Intensity Exposure CARPENTER EXHIBIT B

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(Cell Tower, Wi-Fi, Wireless Laptop and 'Smart' Meter RF Intensities)

Ray, 1990 Ilses Seaman, 1999	A 1-hr cell phone exposure causes chromatin condensation; impaired DNA repair mechanisms; last 3 days (longer than stress response) the effect reaches saturation in only one hour of exposure; electro- sensitive (ES)	
Ray, 1990	Hyperactivity caused by nitric oxide synthase inhibitor is countered by exposure to ultra-wide band pulses (600/sec) for 30 min	0.037 W/Kg
	Decrease in eating and drinking behavior	0.0317 W/Kg
ne digital Ivaschuk, 1997	Activity of c-jun (oncogene or cancer gene) was altered in cells after 20 minutes exposure to cell phone digital TDMA signal	0.026 W/Kg
age) and still leaking Salford, 2003	A single, 2-hr exposure to GSM cell phone radiation results in serious neuron damage (brain cell damage) and death in cortex, hippocampus, and basal ganglia of brain- even 50+ days later blood-brain barrier is still leaking albumin (P<.002) following only one cell phone exposure	0.02 W/Kg
Veyret, 1991	Immune system effects - elevation of PFC count (antibody-producing cells)	0.015 W/Kg
s Kumar, 2012	Sperm damage from oxidative stress and lowered melatonin levels resulted from 2-hr per day/45 days exposure to 10 GHz.	0.014 W/Kg
nay be Stagg, 1997	Cell phone RFR induces glioma (brain cancer) cells to significantly increase thymidine uptake, which may be indication of more cell division	0.0059 W/Kg
and diation at Persson, 1997 man	915 MHz cell phone RFR caused pathological leakage of blood-brain barrier. Worst at lower SAR levels and worse with CW compared to Frequency of pathological changes was 35% in rats exposed to pulsed radiation at 50% to continuous wave RFR. Effects observed at a specific absorption (SA) of > 1.5 joules/Kg in human tissues	0.004 - 0.008 W/Kg
stress Velizarov, 1999	A significant change in cell proliferation not attributable to thermal heating. RFR induces non-thermal stress proteins (960 MHz GSM)	0.0021 W/Kg
KFR (900 Wolke, 1996	Statistically significant change in intracellular calcium concentration in heart muscle cells exposed to RFR (900 MHz/50 Hz modulation)	0.001 W/Kg
Reference		SAR (Watts/Kilogram)

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effects Sk	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure Ca	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism Ca	Cardiac, heart muscle, blood-pressure, vascular effects

SAR (Watts/Kilogram)		Reference
0.05 W/Kg	Significant increase in firing rate of neurons (350%) with pulsed 900 MHz cell phone radiation exposure (but not with CW) in avian brain cells	Beason, 2002
0.09 W/Kg	900 MHz study of mice for 7 days, 12-hr per day (whole-body) resulted in significant effect on mitochondria and genome stability	Aitken, 2005
0.091 W/Kg	Wireless internet 2400 MHz, 24-hrs per day/20 weeks increased DNA damage and reduced DNA repair; levels below 802.11 g Authors say "findings raise questions about safety of radiofrequency exposure from Wi-Fi internet access devices for growing organisms of reproductive age, with a potential effect on fertility and integrity of germ cells" (male germ cells are the reproductive cells=sperm)	Atasoy, 2012
0.11 W/Kg	Increased cell death (apoptosis) and DNA fragmentation at $2.45~\mathrm{GHz}$ for $35~\mathrm{days}$ exposure (chronic exposure study)	Kesari, 2010
0.121 W/Kg	Cardiovascular system shows significant decrease in arterial blood pressure (hypotension) after exposure to ultra-wide band pulses	Lu, 1999
0.13 - 1.4 W/Kg	Lymphoma cancer rate doubled with two 1/2-hr exposures per day of cell phone radiation for 18 months (pulsed 900 MHz cell signal)	Repacholi, 1997
0.14 W/Kg	Elevation of immune response to RFR exposure	Elekes, 1996
0.141 W/Kg	Structural changes in testes - smaller diameter of seminiferous	Dasdag, 1999
0.15 - 0.4 W/Kg	Statistically significant increase in malignant tumors in rats chronically exposed to RFR	Chou, 1992
0.26 W/Kg	Harmful effects to the eye/certain drugs sensitize the eye to RFR	Kues, 1992
0.28 - 1.33 W/Kg	Significant increase in reported headaches with increasing use of hand-held cell phone use (maximum tested was 60 min per day)	Chia, 2000
0.3 - 0.44 W/Kg	Cell phone use results in changes in cognitive thinking/mental tasks related to memory retrieval	Krause, 2000
0.3 - 0.44 W/Kg	Attention function of brain and brain responses are speeded up	Preece, 1999
0.3 - 0.46 W/Kg	Cell phone RFR doubles pathological leakage of blood-brain barrier permeability at two days ($P=.002$) and triples permeability at four days ($P=.001$) at 1800 MHz GSM cell phone radiation	Schirmacher, 2000

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

SAR (Watts/Kilogram)		Reference
0.43 W/Kg	Significant decrease in sperm mobility; drop in sperm concentration; and decrease in seminiferous tubules at 800 MHz, 8-hr/day, 12 weeks, with mobile phone radiation level on STANDBY ONLY (in rabbits)	Salama, 2008
0.5 W/Kg	900 MHz pulsed RF affects firing rate of neurons (Lymnea stagnalis) but continuous wave had no effect	Bolshakov, 1992
0.58 - 0.75 W/Kg	Decrease in brain tumors after chronic exposure to RFR at 836 MHz	Adey, 1999
0.6 - 0.9 W/Kg	Mouse embryos develop fragile cranial bones from in utero 900 MHz The authors say "(O)ur results clearly show that even modest exposure (e.g., 6 min daily for 21 days" is sufficient to interfere with the normal mouse developmental process"	Fragopoulou, 2009
0.6 and 1.2 W/Kg	Increase in DNA single and double-strand DNA breaks in rat brain cells with exposure to 2450 MHz RFR	Lai & Singh, 1996
0.795 W/Kg	GSM 900 MHz, 217 Hz significantly decreases ovarian development and size of ovaries, due to DNA damage and premature cell death of nurse cells and follicles in ovaries (that nourish egg cells)	Panagopoulous, 2012
0.87 W/Kg	Altered human mental performance after exposure to GSM cell phone radiation (900 MHz TDMA digital cell phone signal)	Hamblin, 2004
0.87 W/Kg	Change in human brainwaves; decrease in EEG potential and statistically significant change in alpha (8-13 Hz) and beta (13-22 Hz) brainwave activity in humans at 900 MHz; exposures 6/min per day for 21 days (chronic exposure)	D'Costa, 2003
0.9 W/Kg	Decreased sperm count and more sperm cell death (apoptosis) after 35 days exposure, 2-hr per day	Kesari, 2012
< 1.0 W/Kg	Rats exposed to mobile phone radiation on STANDBY ONLY for 11-hr 45-min plus 15-min TRANSMIT mode; 2 times per day for 21 days showed decreased number of ovarian follicles in pups born to these pregnant rats. The authors conclude "the decreased number of follicles in pups exposed to mobile phone microwaves suggest that intrauterine exposure has toxic effects on ovaries."	Gul, 2009
0.4 - 1.0 W/Kg	One 6-hr exposure to 1800 MHz cell phone radiation in human sperm cells caused a significant dose response and reduced sperm motility and viability; reactive oxygen species levels were significantly increased after exposure to 1.0 W/Kg; study confirms detrimental effects of RF/MW to human sperm. The authors conclude "(T)hese findings have clear implications for the safety of extensive mobile phone use by males of reproductive age, potentially affecting both their fertility and the health and wellbeing of their offspring."	De Iuliis, 2009

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

SAR (Watts/Kilogram)		Reference
1.0 W/Kg	Human semen degraded by exposure to cell phone frequency RF increased free-radical damage.	De Iuliis, 2009
1.0 W/Kg	Motility, sperm count, sperm morphology, and viability reduced in active cell phone users (human males) in dose-dependent manner.	Agarwal, 2008
1.0 W/Kg	GSM cell phone use modulates brain wave oscillations and sleep EEG	Huber, 2002
1.0 W/Kg	Cell phone RFR during waking hours affects brain wave activity. (EEG patterns) during subsequent sleep	Achermann, 2000
1.0 W/Kg	Cell phone use causes nitric oxide (NO) nasal vasodilation (swelling inside nasal passage) on side of head phone use	Paredi, 2001
1.0 W/Kg	Four-fold increase in eye cancer (uveal melanoma) in cell phone users	Stang, 2001
1.0 W/Kg	Increase in headache, fatigue and heating behind ear in cell phone users	Sandstrom, 2001
1.0 W/Kg	Significant increase in concentration difficulties using 1800 MHz cell phone compared to 900 MHz cell phone	Santini, 2001
1.0 W/Kg	Sleep patterns and brain wave activity are changed with 900 MHz cell phone radiation exposure during sleep	Borbely, 1999
1.4 W/Kg	GSM cell phone exposure induced heat shock protein HSP 70 by 360% (stress response) and phosphorylation of ELK-1 by 390%	Weisbrot, 2003
1.46 W/Kg	850 MHz cell phone radiation decreases sperm motility, viability is significantly decreased; increased oxidative damage (free-radicals) significantly decreased; increased oxidative damage (free-radicals)	Agarwal, 2009
1.48 W/Kg	A significant decrease in protein kinase C activity at 112 MHz with 2-hr per day for 35 days; hippocampus is site, consistent with reports that RFR negatively affects learning and memory functions	Paulraj, 2004
1.0 - 2.0 W/Kg	Significant elevation in micronuclei in peripheral blood cells at 2450 MHz (8 treatments of 2-hr each)	Trosic, 2002
1.5 W/Kg	GSM cell phone exposure affected gene expression levels in tumor suppressor p53-deficient embryonic stem cells; and significantly increased HSP 70 heat shock protein production	Czyz, 2004

Suress proceins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

CARPENTER EXHIBIT B

SAR (Watts/Kilogram)		Reference
1.8 W/Kg	Whole-body exposure to RF cell phone radiation of 900-1800 MHz 1 cm from head of rats caused high incidence of sperm cell death; deformation of sperm cells; prominent clumping together of sperm cells into "grass bundle shapes" that are unable to separate/swim. Sperm cells unable to swim and fertilize in normal manner.	Yan, 2007
2.0 W/Kg	GSM cell phone exposure of 1-hr activated heat shock protein HSP 27 (stress response) and P38 MAPK (mutagen-activated protein kinase) that authors say facilitates brain cancer and increased blood-brain barrier permeability, allowing toxins to cross BBB into brain	Leszczynski, 2002
2 W/Kg	900 MHz cell phone exposure caused brain cell oxidative damage by increasing levels of NO, MDA, XO and ADA in brain cells; caused statistically significant increase in 'dark neurons' or damaged brain cells in cortex, hippocampus and basal ganglia with a 1-hr exposure for 7 consecutive days	Ilhan, 2004
2.6 W/Kg	900 MHz cell phone exposure for 1-hr significantly altered protein expression levels in 38 proteins following irradiation; activates P38 MAP kinase stress signalling pathway and leads to changes in cell sie and shape (shrinking and rounding up) and to activation of HSP 27, a stress protein (heat shock protein)	Leszczynski, 2004
2.0 - 3.0 W/Kg	RFR accelerated development of both skin and breast tumors	Szmigielski, 1982
2 W/Kg	Pulse-modulated RFR and MF affect brain physiology (sleep study)	Schmidt, 2012

STANDARDS		
0.08 W/Kg	IEEE Standard uncontrolled public environment (whole body)	IEEE
0.4 W/Kg	IEEE Standard controlled occupational environment (whole body)	IEEE
1.6 W/Kg	FCC (IEEE) SAR limit for 1 gram of tissue in a partial body exposure	FCC, 1996
2 W/Kg	ICNIRP SAR limit for 10 grams of tissue	ICNIRP, 1996

Cardiac, heart muscle, blood-pressure, vascular effects	Disrupted calcium metabolism
Cancer (other than brain), cell proliferation	Oxidative damage/ROS/DNA damage/DNA repair failure
Sleep, neuron firing rate, EEG, memory, learning, behavior	Reproduction/fertility effeccts
Brain tumors and blood-brain barrier	Stress proteins, HSP, disrupted immune function

Reference List Reported Biological Effects from Radiofrequency Radiation (RFR) at Low-Intensity Exposure Levels

(Cell Tower, WI-FI, Wireless Laptop, Wireless Utility Meters 'smart meters')

Prepared November 22, 2012 by: Cindy Sage. MA, Sage Associates

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Case 2:20-cv-00237-JCN Document 137-14 Filed 06/13/24 Page 112 of 138 PageID #: 3716 CARPENTER EXHIBIT B

Reported Biological Effects from Radiofrequency Radiation at Low-Intensity Exposure (Cell Tower, Wi-Fi, Wireless Laptop and 'Smart' Meter RF Intensities)

CARPENTER DEPO EXHIBIT 5

Power Density (Microwatts/centime	eter2 - uW/cm2)	Reference
As low as (10 ⁻¹³) or 100 femtowatts/cm2	Super-low intensity RFR effects at MW reasonant frequencies resulted in changes in genes; problems with chromatin conformation (DNA)	Belyaev, 1997
5 picowatts/cm2 (10- 12)	Changed growth rates in yeast cells	Grundler, 1992
0.1 nanowatt/cm2 (10- ¹⁰) or 100 picowatts/cm2	Super-low intensity RFR effects at MW reasonant frequencies resulted in changes in genes; problems with chromatin condensation (DNA) intensities comparable to base stations	Belyaev, 1997
0.00034 uW/cm2	Chronic exposure to mobile phone pulsed RF significantly reduced sperm count,	Behari, 2006
0.0005 uW/cm2	RFR decreased cell proliferation at 960 MHz GSM 217 Hz for 30-min exposure	Velizarov, 1999
0.0006 - 0.0128 uW/cm2	Fatigue, depressive tendency, sleeping disorders, concentration difficulties, cardio- vascular problems reported with exposure to GSM 900/1800 MHz cell phone signal at base station level exposures.	Oberfeld, 2004
0.0009 uW/cm2	RFR induced 10%-40% increase in DNA synthesis in glioma cells (brain)	Stagg, 1997
0.003 - 0.02 uW/cm2	In children and adolescents (8-17 yrs) short-term exposure caused headache, irritation, concentration difficulties in school.	Heinrich, 2010
0.003 to 0.05 uW/cm2	In children and adolescents (8-17 yrs) short-term exposure caused conduct problems in school (behavioral problems)	Thomas, 2010
0.005 uW/cm2	In adults (30-60 yrs) chronic exposure caused sleep disturbances, (but not significantly increased across the entire population)	Mohler, 2010
0.005 - 0.04 uW/cm2	Adults exposed to short-term cell phone radiation reported headaches, concentration difficulties (differences not significant, but elevated)	Thomas, 2008
0.006 - 0.01 uW/cm2	Chronic exposure to base station RF (whole-body) in humans showed increased stress hormones; dopamine levels substantially decreased; higher levels of adrenaline and nor-adrenaline; dose-response seen; produced chronic physiological stress in cells even after 1.5 years.	Buchner, 2012
0.01 - 0.11 uW/cm2	RFR from cell towers caused fatigue, headaches, sleeping problems	Navarro, 2003

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier		
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior		
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation		
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects		

Power Density (Microwatts/centime	eter2 - uW/cm2)	Reference
0.01 - 0.05 uW/cm2	Adults (18-91 yrs) with short-term exposure to GSM cell phone radiation reported headache, neurological problems, sleep and concentration problems.	Hutter, 2006
0.005 - 0.04 uW/cm2	Adults exposed to short-term cell phone radiation reported headaches, concentration difficulties (differences not significant, but elevated)	Thomas, 2008
0.015 - 0.21 uW/cm2	Adults exposed to short-term GSM 900 radiation reported changes in mental state (e.g., calmness) but limitations of study on language descriptors prevented refined word choices (stupified, zoned-out)	Augner, 2009
0.05 - 0.1 uW/cm2	RFR linked to adverse neurological, cardio symptoms and cancer risk	Khurana, 2010
0.05 - 0.1 uW/cm2	RFR related to headache, concentration and sleeping problems, fatigue	Kundi, 2009
0.07 - 0.1 uW/cm2	Sperm head abnormalities in mice exposed for 6-months to base station level RF/MW. Sperm head abnormalities occurred in 39% to 46% exposed mice (only 2% in controls) abnormalities was also found to be dose dependent. The implications of the pin-head and banana-shaped sperm head. The occurrence of sperm head observed increase occurrence of sperm head abnormalities on the reproductive health of humans living in close proximity to GSM base stations were discussed."	Otitoloju, 2010
0.38 uW/cm2	RFR affected calcium metabolism in heart cells	Schwartz, 1990
0.8 - 10 uW/cm2	RFR caused emotional behavior changes, free-radical damage by super-weak MWs	Akoev, 2002
0.13 uW/cm2	RFR from 3G cell towers decreased cognition, well-being	Zwamborn, 2003
0.16 uW/cm2	Motor function, memory and attention of school children affected (Latvia)	Kolodynski, 1996
0.168 - 1.053 uW/cm2	Irreversible infertility in mice after 5 generations of exposure to RFR from an 'antenna park'	Magras & Zenos, 1997
0.2 - 8 uW/cm2	RFR caused a two-fold increase in leukemia in children	Hocking, 1996
0.2 - 8 uW/cm2	RFR decreased survival in children with leukemia	Hocking, 2000
0.21 - 1.28 uW/cm2	Adolescents and adults exposed only 45 min to UMTS cell phone radiation reported increases In headaches.	Riddervold, 2008

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart-muscle, blood-pressure, vascular effects	

Power Density (Microwatts/centin	neter2 - uW/cm2)	Reference
0.5 uW/cm2	Significant degeneration of seminiferous epithelium in mice at 2.45 GHz, 30-40 min.	Saunders, 1981
0.5 - 1.0 uW/cm2	Wi-FI level laptop exposure for 4-hr resulted in decrease in sperm viability, DNA fragmentation with sperm samples placed in petri dishes under a laptop connected via WI-FI to the internet.	Avendano, 2012
1.0 uW/cm2	RFR induced pathological leakage of the blood-brain barrier	Persson, 1997
1.0 uW/cm2	RFR caused significant effect on immune function in mice	Fesenko, 1999
1.0 uW/cm2	RFR affected function of the immune system	Novoselova, 1999
1.0 uW/cm2	Short-term (50 min) exposure in electrosensitive patients, caused loss of well-being after GSM and especially UMTS cell phone radiation exposure	Eltiti, 2007
1.3 - 5.7 uW/cm2	RFR associated with a doubling of leukemia in adults	Dolk, 1997
1.25 uW/cm2	RFR exposure affected kidney development in rats (in-utero exposure)	Pyrpasopoulou, 2004
1.5 uW/cm2	RFR reduced memory function in rats	Nittby, 2007
2 uW/cm2	RFR induced double-strand DNA damage in rat brain cells	Kesari, 2008
2.5 uW/cm2	RFR affected calcium concentrations in heart muscle cells	Wolke, 1996
2 - 4 uW/cm2	Altered cell membranes; acetycholine-induced ion channel disruption	D'Inzeo, 1988
4 uW/cm2	RFR caused changes in hippocampus (brain memory and learning)	Tattersall, 2001
4 - 15 uW/cm2	Memory impairment, slowed motor skills and retarded learning in children	Chiang, 1989
5 uW/cm2	RFR caused drop in NK lymphocytes (immune function decreased)	Boscolo, 2001
5.25 uW/cm2	20 minutes of RFR at cell tower frequencies induced cell stress response	Kwee, 2001
5 - 10 uW/cm2	RFR caused impaired nervous system activity	Dumansky, 1974
6 uW/cm2	RFR induced DNA damage in cells	Phillips, 1998

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects	

Power Density (Microwatts/centin	neter2 - uW/cm2)	Reference
8.75 uW/cm2	RFR at 900 MHz for 2-12 hours caused DNA breaks in leukemia cells	Marinelli, 2004
10 uW/cm2	Changes in behavior (avoidance) after 0.5 hour exposure to pulsed RFR	Navakatikian, 1994
10 - 100 uW/cm2	Increased risk in radar operators of cancer; very short latency period; dose response to exposure level of RFR reported.	Richter, 2000
12.5 uW/cm2	RFR caused calcium efflux in cells - can affect many critical cell functions	Dutta, 1989
13.5 uW/cm2	RFR affected human lymphocytes - induced stress response in cells	Sarimov, 2004
14.75 uW/cm2	RFR increased biomarker for cell division in glioma brain tumor cells	Stagg, 1997
20 uW/cm2	Increase in serum cortisol (a stress hormone)	Mann, 1998
28.2 uW/cm2	RFR increased free radical production in rat cells	Yurekli, 2006
37.5 uW/cm2	Immune system effects - elevation of PFC count (antibody producing cells	Veyret, 1991
45 uW/cm2	Pulsed RFR affected serum testosterone levels in mice	Forgacs, 2006
50 uW/cm2	Cell phone RFR caused a pathological leakage of the blood-brain barrier in 1 hour	Salford, 2003
50 uW/cm2	An 18% reduction in REM sleep (important to memory and learning functions)	Mann, 1996
60 uW/cm2	RFR caused structural changes in cells of mouse embryos	Somozy, 1991
60 uW/cm2	Pulsed RFR affected immune function in white blood cells	Stankiewicz, 2006
60 uW/cm2	Cortex of the brain was activated by 15 minutes of 902 MHz cell phone	Lebedeva, 2000
65 uW/cm2	RFR affected genes related to cancer	Ivaschuk, 1999
92.5 uW/cm2	RFR caused genetic changes in human white blood cells	Belyaev, 2005
100 uW/cm2	Changes in immune function	Elekes, 1996
100 uW/cm2	A 24.3% drop in testosterone after 6 hours of CW RFR exposure	Navakatikian, 1994

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

Power Density (Microwatts/centimeter2 - uW/cm2)		Reference
120 uW/cm2	A pathological leakage in the blood-brain barrier with 915 MHz cell RF	Salford, 1994
500 uW/cm2	Intestinal epithelial cells exposed to 2.45 GHz pulsed at 16 Hz showed changes in intercellular calcium.	Somozy, 1993
500 uW/cm2	A 24.6% drop in testosterone and 23.2% drop in insulin after 12 hrs of pulsed RFR exposure.	Navakatikian, 1994

STANDARDS		
530 - 600 uW/cm2	Limit for uncontrolled public exposure to 800-900 MHz	ANSI/IEEE and FCC
1000 uW/cm2	PCS STANDARD for public exposure (as of September 1,1997)	FCC, 1996
5000 uW/cm2	PCS STANDARD for occupational exposure (as of September 1, 1997)	FCC, 1996
BACKGROUND LEVE	LS	
0.003 uW/cm2	Background RF levels in US cities and suburbs in the 1990s	Mantiply, 1997
0.05 uW/cm2	Median ambient power density in cities in Sweden (30-2000 MHz)	Hamnierius, 2000
0.1 - 10 uW/cm2	Ambient power density within 100-200' of cell site in US (data from 2000)	Sage, 2000

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

SAR (Watts/Kilogram)		Reference
0.000064 - 0.000078 W/Kg	Well-being and cognitive function affected in humans exposed to GSM-UMTS cell phone frequencies; RF levels similar near cell sites	TNO Physics and
0.00015 - 0.003 W/Kg	. Calcium ion movement in isolated frog heart tissue is increased 18% ((P<.01)) and by 21% (P<.05) by weak RF field modulated at 16 Hz	Schwartz, 1990
0.000021 - 0.0021 W/Kg	Changes in cell cycle; cell proliferation (960 MHz GSM mobile phone)	Kwee, 1997
0.0003 - 0.06 W/Kg	Neurobehavioral disorders in offspring of pregnant mice exposed in utero to cell phones - dose-response impaired glutamatergic synaptic transmission onto layer V pyramidal neurons of the prefrontal cortex. Hyperactivity and impaired memory function in offspring. Altered brain development.	Aldad, 2012
0.0009 W/Kg	Changes in brain glial cells with TDMA 836.55 MHz frequency	Stagg, 1997
0.0016 - 0.0044 W/Kg	Very low power 700 MHz CW affects excitability of hippocampus tissue, consistent with reported behavioral changes.	Tattersall, 2001
0.0021 W/Kg	Heat shock protein HSP 70 is activated by very low intensity microwave exposure in human epithelial amnion cells	Kwee, 2001
0.0024 - 0.024 W/Kg	Digital cell phone RFR at very low intensities causes DNA damage in human cells; both DNA damage and impairment of DNA is reported	Phillips, 1998
0.0027 W/Kg	Changes in active avoidance conditioned behavioral effect is seen after one-half hour of pulsed radiofrequency radiation	Navakatikian, 1994
0.0035 W/Kg	900 MHz cell phone signal induces DNA breaks and early activation of p53 gene; short exposure of 2-12 hours leads cells to acquire greater survival chance - linked to tumor agressiveness.	Marinelli, 2004
0.0095 W/Kg	MW modulated at 7 Hz produces more errors in short-term memory function on complex tasks (can affect cognitive processes such as attention and memory)	Lass, 2002
0.001 W/Kg	750 MHz continuous wave (CW) RFR exposure caused increase in heat shock protein (stress proteins). Equivalent to what would be induced by 3 degree C. heating of tissue (but no heating occurred)	De Pomerai, 2000

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle; blood-pressure, vascular effects

SAR (Watts/Kilogram)		Reference	
0.001 W/Kg	Statistically significant change in intracellular calcium concentration in heart muscle cells exposed to RFR (900 MHz/50 Hz modulation)	Wolke, 1996	
0.0021 W/Kg	A significant change in cell proliferation not attributable to thermal heating. RFR induces non-thermal stress proteins (960 MHz GSM)	S Velizarov, 1999	
0.004 - 0.008 W/Kg	915 MHz cell phone RFR caused pathological leakage of blood-brain barrier. Worst at lower SAR levels and worse with CW compared to Frequency of pathological changes was 35% in rats exposed to pulsed radiation at 50% to continuous wave RFR. Effects observed at a specific absorption (SA) of > 1.5 joules/Kg in human Persson, 199 tissues		
0.0059 W/Kg	Cell phone RFR induces glioma (brain cancer) cells to significantly increase thymidine uptake, which may be indication of more cell division	Stagg, 1997	
0.014 W/Kg	Sperm damage from oxidative stress and lowered melatonin levels resulted from 2-hr per day/45 days Exposure to 10 GHz. Kumar, 20		
0.015 W/Kg	15 W/Kg Immune system effects - elevation of PFC count (antibody-producing cells) Veyret, 199		
A single, 2-hr exposure to GSM cell phone radiation results in serious neuron damage (brain cell damage) and 0.02 W/Kg death in cortex, hippocampus, and basal ganglia of brain- even 50+ days later blood-brain barrier is still leaking Salford, 20 albumin (P<.002) following only one cell phone exposure		Salford, 2003	
0.026 W/Kg	Activity of c-jun (oncogene or cancer gene) was altered in cells after 20 minutes exposure to cell phone digital TDMA signal	Ivaschuk, 1997	
0.0317 W/Kg	Decrease in eating and drinking behavior	Ray, 1990	
0.037 W/Kg Hyperactivity caused by nitric oxide synthase inhibitor is countered by exposure to ultra-wide band pulses Seaman,		Seaman, 1999	
0.037 - 0.040 W/Kg	A 1-hr cell phone exposure causes chromatin condensation; impaired DNA repair mechanisms; last 3 days (longer than stress response) the effect reaches saturation in only one hour of exposure; electro- sensitive (ES) people have different response in formation of DNA repair foci, compared to healthy individuals; effects depend on carrier frequency (915 MHz = 0.037 W/Kg but 1947 MHz = 0.040 W/Kg)	aturation in only one hour of exposure; electro- sensitive (ES) A repair foci, compared to healthy individuals; effects depend Belyaev, 2008	

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects	

SAR (Watts/Kilogram)		Reference
0.05 W/Kg	Significant increase in firing rate of neurops (350%) with pulsed 900 MHz cell phone radiation exposure (but not with GW) in avian brain cells	Beason, 2002
0.09 W/Kg	900 MHz study of mice for 7 days, 12-hr per day (whole-body) resulted in significant effect on mitochondria and genome stability	Aitken, 2005
0.091 W/Kg	Wireless internet 2400 MHz, 24-hrs per day/20 weeks increased DNA damage and reduced DNA repair; levels below 802.11 g Authors say "findings raise questions about safety of radiofrequency exposure from Wi-Fi internet access devices for growing organisms of reproductive age, with a potential effect on fertility and integrity of germ cells" (male germ cells are the reproductive cells=sperm)	Atasoy, 2012
0.11 W/Kg	Increased cell death (apoptosis) and DNA fragmentation at 2.45 GHz for 35 days exposure (chronic exposure study)	Kesari, 2010
0.121 W/Kg	Gardiovascular system shows significant decrease in arterial blood pressure (hypotension) after exposure to ultra-wide band pulses	Lu. 199 9
0.13 - 1.4 W/Kg	Lymphoma cancer rate doubled with two 1/2-hr exposures per day of cell phone radiation for 18 months (pulsed 900 MHz cell signal)	Repacholi, 1997
0.14 W/Kg	Elevation of immune response to RFR exposure	Elekes, 1996
0.141 W/Kg	Structural changes in testes - smaller diameter of seminiferous	Dasdag, 1999
0.15 - 0.4 W/Kg	Statistically significant increase in malignant tumors in rats chronically exposed to RFR	Chou, 1992
0.26 W/Kg	Harmful effects to the eye/certain drugs sensitize the eye to RFR	Kues, 1992
0.28 - 1.33 W/Kg	Significant increase in reported headaches with increasing use of hand-held cell phone use (maximum tested was 60 min per day)	Chia, 2000
0.3 - 0.44 W/Kg	Cell phone use results in changes in cognitive thinking/mental tasks related to memory retrieval	Krause, 2000
0.3 - 0.44 W/Kg	Attention function of brain and brain responses are speeded up	Preece, 1999
0.3 - 0.46 W/Kg	Cell phone RFR doubles pathological leakage of blood-brain barrier permeability at two days (P=.002) and triples permeability at four days (P=.001) at 1800 MHz GSM cell phone radiation	Schirmacher, 2000

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects	

SAR (Watts/Kilogram)		Reference
0.43 W/Kg	Significant decrease in sperm mobility; drop in sperm concentration; and decrease in seminiferous tubules at 800 MHz, 8-hr/day, 12 weeks, with mobile phone radiation level on STANDBY ONLY (in rabbits)	Salama, 2008
0.5 W/Kg	900 MHz pulsed RF affects firing rate of neurons (Lymnea stagnalis) but continuous wave had no effect	Bolshakov, 1992
0.58 - 0.75 W/Kg	Decrease in brain tumors after chronic exposure to RFR at 836 MHz	Adey, 1999
0.6 - 0.9 W/Kg	Mouse embryos develop fragile cranial bones from in utero 900 MHz The authors say "(O)ur results clearly show that even modest exposure (e.g., 6 min daily for 21 days" is sufficient to interfere with the normal mouse developmental process"	Fragopoulou, 2009
0.6 and 1.2 W/Kg	Increase in DNA single and double-strand DNA breaks in rat brain cells with exposure to 2450 MHz RFR	Lai & Singh, 1996
0.795 W/Kg	GSM 900 MHz, 217 Hz significantly decreases ovarian development and size of ovaries, due to DNA damage and premature cell death of nurse cells and follicles in ovaries (that nourish egg cells)	
0.87 W/Kg Altered human mental performance after exposure to GSM cell phone radiation (900 MHz TDMA digital cell Hamble phone signal)		Hamblin, 2004
0.87 W/Kg	ange in human brainwaves; decrease in EEG potential and statistically significant change in alpha (8-13 Hz) d beta (13-22 Hz) brainwave activity in humans at 900 MHz; exposures 6/min per day for 21 days (chronic D'Costa, 2003 posure)	
0.9 W/Kg	Decreased sperm count and more sperm cell death (apoptosis) after 35 days exposure, 2-hr per day	Kesari, 2012
< 1.0 W/Kg	Rats exposed to mobile phone radiation on STANDBY ONLY for 11-hr 45-min plus 15-min TRANSMIT mode; 2 times per day for 21 days showed decreased number of ovarian follicles in pups born to these pregnant rats. The authors conclude "the decreased number of follicles in pups exposed to mobile phone microwaves suggest that intrauterine exposure has toxic effects on ovaries."	Gul, 2009
0.4 - 1.0 W/Kg	One 6-hr exposure to 1800 MHz cell phone radiation in human sperm cells caused a significant dose response and reduced sperm motility and viability; reactive oxygen species levels were significantly increased after exposure to 1.0 W/Kg; study confirms detrimental effects of RF/MW to human sperm. The authors conclude "(T)hese findings have clear implications for the safety of extensive mobile phone use by males of reproductive age, potentially affecting both their fertility and the health and wellbeing of their offspring."	De Iuliis, 2009

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects	

SAR (Watts/Kilogram)		Reference
1.0 W/Kg	1.0 W/Kg Human semen degraded by exposure to cell phone frequency RF increased free-radical damage. De Iu	
1.0 W/Kg	Motility, sperm count, sperm morphology, and viability reduced in active cell phone users (human males) in dose-dependent manner. Agarwal, 2008	
1.0 W/Kg	GSM cell phone use modulates brain wave oscillations and sleep EEG	Huber, 2002
1.0 W/Kg	Cell phone RFR during waking hours affects brain wave activity. (EEG patterns) during subsequent sleep	Achermann, 2000
1.0 W/Kg	Cell phone use causes nitric oxide (NO) nasal vasodilation (swelling inside nasal passage) on side of head phone use	Paredi, 2001
1.0 W/Kg	Four-fold increase in eye cancer (uveal melanoma) in cell phone users	Stang, 2001
1.0 W/Kg	0 W/Kg Increase in headache, fatigue and heating behind ear in cell phone users Sandstron	
1.0 W/Kg	0 W/Kg Significant increase in concentration difficulties using 1800 MHz cell phone compared to 900 MHz cell phone Santini, 20	
1.0 W/Kg	Sleep patterns and brain wave activity are changed with 900 MHz cell phone radiation exposure during sleep Borbely, 19	
1.4 W/Kg	GSM cell phone exposure induced heat shock protein HSP 70 by 360% (stress response) and phosphorylation of Weisbrot, 2 ELK-1 by 390%	
1.46 W/Kg	850 MHz cell phone radiation decreases sperm motility, viability is significantly decreased; increased oxidative damage (free-radicals) significantly decreased; increased oxidative damage (free-radicals)	Agarwal, 2009
1.48 W/Kg	A significant decrease in protein kinase C activity at 112 MHz with 2-hr per day for 35 days; hippocampus is site, consistent with reports that RFR negatively affects learning and memory functions	Paulraj, 2004
1.0 - 2.0 W/Kg	/Kg Significant elevation in micronuclei in peripheral blood cells at 2450 MHz (8 treatments of 2-hr each) Trosic, 2002	
1.5 W/Kg	GSM cell phone exposure affected gene expression levels in tumor suppressor p53-deficient embryonic stem cells; and significantly increased HSP 70 heat shock protein production	Czyz, 2004

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier	
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior	
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation	
Disrupted calcium metabolism	Cardiac, heart muscle, blood pressure, vascular effects	

SAR (Watts/Kilogram)		Reference
1.8 W/Kg	Whole-body exposure to RF cell phone radiation of 900-1800 MHz 1 cm from head of rats caused high incidence of sperm cell death; deformation of sperm cells; prominent clumping together of sperm cells into "grass bundle shapes" that are unable to separate/swim. Sperm cells unable to swim and fertilize in normal manner.	Yan, 2007
2.0 W/Kg	GSM cell phone exposure of 1-hr activated heat shock protein HSP 27 (stress response) and P38 MAPK (mutagen-activated protein kinase) that authors say facilitates brain cancer and increased blood-brain barrier permeability, allowing toxins to cross BBB into brain	Leszczynski, 2002
2 W/Kg	900 MHz cell phone exposure caused brain cell oxidative damage by increasing levels of NO, MDA, XO and ADA in brain cells; caused statistically significant increase in 'dark neurons' or damaged brain cells in cortex, hippocampus and basal ganglia with a 1-hr exposure for 7 consecutive days	Ilhan, 2004
2.6 W/Kg	900 MHz cell phone exposure for 1-hr significantly altered protein expression levels in 38 proteins following irradiation; activates P38 MAP kinase stress signalling pathway and leads to changes in cell sie and shape (shrinking and rounding up) and to activation of HSP 27, a stress protein (heat shock protein)	Leszczynski, 2004
2.0 - 3.0 W/Kg	RFR accelerated development of both skin and breast tumors	Szmigielski, 1982
2 W/Kg	Pulse-modulated RFR and MF affect brain physiology (sleep study)	Schmidt, 2012

STANDARDS		
0.08 W/Kg	IEEE Standard uncontrolled public environment (whole body)	IEEE
0.4 W/Kg	IEEE Standard controlled occupational environment (whole body)	IEEE
1.6 W/Kg	FCC (IEEE) SAR limit for 1 gram of tissue in a partial body exposure	FCC, 1996
2 W/Kg	ICNIRP SAR limit for 10 grams of tissue	ICNIRP, 1996

Stress proteins, HSP, disrupted immune function	Brain tumors and blood-brain barrier
Reproduction/fertility effeccts	Sleep, neuron firing rate, EEG, memory, learning, behavior
Oxidative damage/ROS/DNA damage/DNA repair failure	Cancer (other than brain), cell proliferation
Disrupted calcium metabolism	Cardiac, heart muscle, blood-pressure, vascular effects

CARPENTER DEPO EXHIBIT 6

Reference List

Reported Biological Effects from Radiofrequency Radiation (RFR) at Low-Intensity Exposure Levels

(Cell Tower, WI-FI, Wireless Laptop, Wireless Utility Meters 'smart meters')

Prepared November 22, 2012 by: Cindy Sage. MA, Sage Associates

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EXPERT REPORT

CARPENTER DEPO EXHIBIT 7

Re: Friedman vs. Central Maine Power Company

2:20-cv-00237-JDL

David O. Carpenter, MD

Qualifications:

My name is David O. Carpenter. I am a public health physician and professor, with a medical degree from Harvard Medical School. My current title is Director of the Institute for Health and the Environment at the University at Albany, which has been designed as a Collaborating Centre of the World Health Organization, as well as Professor of Environmental Health Sciences within the School of Public Health. In addition I am an Honorary Professor, Queensland Children's Medical Research Unit, University of Queensland, Brisbane, Australia. Formerly, I was the Director of the Wadsworth Center for Laboratories and Research of the New York State Department of Health (1980-1985) and the Dean of the School of Public Health at the University of Albany (1985-1998), while remaining employed by the New York State Department of Health. I assumed my current position in 1998.

In the 1980s I served as Executive Secretary of the New York State Powerlines Project, a state-funded study designed to determine whether there were adverse health effects from living near power lines. The program of research showed children living in homes with elevated magnetic fields coming from power lines suffered from an elevated risk of developing leukemia, and that electromagnetic field (EMF) exposure altered a variety of responses studied in animals and in cellular systems. After the Powerlines Project was finished, I became the spokesperson for New York State on the issue of health effects of electromagnetic fields.

I have published several reviews and have edited two books on the <u>Biologic Effects of Electric and Magnetic Fields</u>. I am also a Co-Editor and a Contributing Author of the *BioInitiative Report: A Rationale for a Biologically-based Public Exposure Standards for Electromagnetic Fields (ELF and RF)* (www.bioinitative.org). This report was first published in 2007, and has been updated in 2012 and 2014. The *BioInitiative Report* documents effects on cellular and animals model systems, adverse human health effects and public health conclusions about impacts of electromagnetic radiation [electromagnetic fields including extremely-low frequency (ELF-EMF) and radiofrequency /microwave (RF/MW-EMF) fields]. I will refer to specific sections of the report where appropriate. I also reference the entire report as a comprehensive and up-to-date review of the scientific information on this subject.

Attached as Exhibit A is my *curriculum vitae*. I am an active researcher and educator. My research activities at present are focused on the general subject of environmental causes of human disease. I have authored over 460 major publications in peer-reviewed scientific journals, have edited six books and have numerous other publications as listed in my *curriculum vitae*. I participate in many international, national, state and local organizations and committees as listed in my *curriculum vitae* along with the Honors, Awards, and Fellowships I have received. I have had sufficient research, administrative and evaluation responsibilities related to human health effects of power line magnetic fields so as to be known as an international expert in this field. In 2009, I was invited to present to the United States President's Cancer Panel on the subject of

power line and radiofrequency fields and cancer. This invitation was based on recognition that I am an expert on this subject. In addition I was invited to testify on the more general issue of adverse health effects resulting from human exposure to electromagnetic fields before the United States House of Representatives. This invitation was based on my reputation as being an expert on this subject. I have also testified on the specific subject of adverse health effects resulting from exposure to magnetic fields arising from power lines. In recent cases I was certified by the courts to be qualified as an expert for health effects of magnetic fields coming from power lines (State of New Jersey, Board of Public Utilities, BPU Docket No. EO16080750; OAL Docket No. PUC 12098-16; 6/22/2018) and radiofrequency electromagnetic fields (US District Court, District of Massachusetts, Civil Action No. 4:15-CV-40116-TSH; 12/8/2015).

This Case:

Mr. Ed Friedman suffers from a form of non-Hodgkin's Lymphoma, called Waldenstrom's macroglobulinemia. This is in general not a curable disease although progression can be delayed with treatment. Mr. Friedman, as well as other residents of Maine, had opposed the installation of RF-emitting smart meters as replacement of analog meters which require someone to read the degree of electrical usage. The courts ruled that Central Maine Power (CMP) could install smart meters, but when an individual requested for any reason, to opt-out of having one placed on their home, they could do so, but would be charged an opt-out fee in addition to the regular electrical usage bill. Mr. Friedman is challenging the right of Central Maine Power to force him to pay surcharges under the American's With Disabilities Act (ADA), Fair Housing Act (FHA) and Rehabilitation Act (RA), based on his cancer, for the same safe power his non-disabled neigbors receive without an extra surcharge. Prior to his disconnection by CMP, he did pay his usage bill. Were they to reconnect him, he requests relief from the discriminatory surcharge because of his cancer. The risk of exacerbating his cancer from 24/7 smart meter exposure means he does not have an option to opt-out as one might with other objections to smart meters but instead has a necessity to avoid the smart meter.

The Americans with Disabilities Act (as well as FHA and RA) are designed to prevent discrimination against individuals who suffer from any disability for which the usual scope of business or other activity would put the disabled person at a disadvantage. The ADA also specifically prohibits imposition of a surcharge on an individual with a disability in order for that individual to avoid the discriminating action. (The FHA specifically cites discrimination in the provision of services and the RA prohibits discrimination for any recipient of federal funding like CMP.) In this case that action is for Mr. Friedman to avoid the likely exacerbating effects of exposure to radiofrequency EMFs that would occur upon placement of a smart meter on his house.

Smart Meters:

There are no careful studies specifically on cancer being caused by electronic meters to my knowledge, in great part because they haven't been around very long, cancer has a long latency between exposure and disease, and all of us are exposed to multiple sources of RF. But smart meter utilize the same type of RF radiation that is used in cell phones. It should be noted that the World Health Organization has declared radiofrequency radiation (including that from smart meters) to be a possible human carcinogen. While it is true that the nature of exposure to RF from electronic meters is only somewhat different from that coming from other wireless devices,

what is important is cumulative, aggregate RF exposure. Most electronic meters transmit signals to the utility for relatively short periods of time but generate radiofrequency pulses at frequent intervals all day and night. Thus the device continuously generates modulated pulses of RF radiation that will expose anyone nearby 24/7. Besides airborne transmission emissions to the utility there is some evidence these RF emissions as well as lower frequency emissions affecting power quality, may be transmitted throughout structures as conducted and radiating emissions on building wiring, acting as an antenna.(Isotrope, 2013).

The waveform used by smart meters consists of very high intensity but very brief burst of pulses. While the average total exposure over periods of time usually do not exceed FCC standards, those standards are not set based on critical peak intensities. An analogy, illustrating the importance of peak is often made with a bullet, the force and damage from the peak impact being far more than the average force. There is building evidence that the rapid rise and fall of these RF peaks is particularly provocative to humans.

While there are not yet specific studies connecting smart meters directly to cancer, there have long been studies showing DNA damage from exposure to RF including at 2450MHz (Lai and Singh 1995), the same frequency used by CMP smart meters. DNA damage is often a precursor to cancers whether through oxidative stress (Yakemenko, et al., 2015) or other mechanisms.

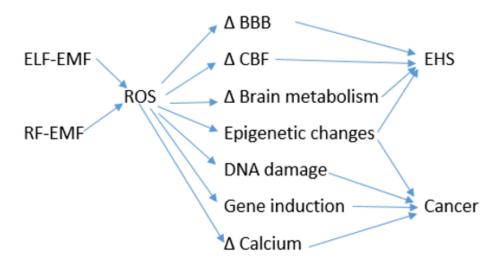
Most of the published reports on health effects specifically from smart meters are directed at the syndrome of electro-hypersensitivity (EHS). This is found in some individuals who in the presence of elevated RF-EMFs develop symptoms of fatigue, headache, brain fog, tinnitus and other symptoms that in some people can be very disabling. EHS symptoms like these and others share much in common with many cancer symptoms and cancer treatment side-effect symptoms so Mr. Friedman's concerns are well founded that RF exposure risks exacerbating some of his present symptoms. These high intensity pulses may be more provocative than continuous waves, as suggested by the report of McCarty et al. (2011), who reported on symptoms experienced by a physician who suffered from EHS, and upon testing in a doubled blinded manner, responded to the on and off of EMFs, not to the steady state.

There is also significant information on self-reported changes in health status by individuals after installation of smart meters on their homes. In a smart meter health effects survey and report submitted and accepted as expert testimony by Dr. Conrad to the Maine PUC in 2013, nearly 98% of the 210 respondents were very sure or fairly sure their new or worsened symptoms correlated with smart meter exposure. 42% of these respondents began suffering symptoms or had preexisting symptoms worsen after smart meter installation but prior to any knowledge the meters had been installed. Frequent symptoms included pressure in head, ringing, buzzing/tinnitus, difficulty concentrating, insomnia, heart racing, arrhythmia, headaches and fatigue. Lamech (2014) published a peer-reviewed study of 142 Australians who submitted information on a public web site on changes in health status after a smart meter was installed on their home. The most frequent symptoms were insomnia, headaches, tinnitus, fatigue and cognitive disturbances. She suggests that these individuals have developed EHS as a result of the smart meter RF EMFs. Carpenter (2015) reported on statements made by a California physician and her husband who had a smart meter placed on their home without their knowledge while they were away from home. Upon returning home both developed the symptoms of EHS, which were only later found to be due to the presence of the smart meter. The symptoms were markedly reduced after the smart meter was removed, although both persons remained at elevated sensitivity to EMFs up until the present.

Radiofrequency fields and cancer:

In 2011 IARC made the designation that exposure to radiofrequency electromagnetic fields was a "possible human carcinogen", based primarily on studies of development of brain cancer among individuals who used mobile phones frequently for 10 years or more. One reason that RF-EMFs were not rated as "probable" or "known" human carcinogens was the lack of evidence that these exposures caused cancer in animals. The has changed since 2011 when both the US National Toxicology Program (Wyde et al., 2018) and a study from the Ramazzini Institute in Italy (Falcioni et al., 2018) have found that RF-EMFs at intensities modeled to reflect whole body exposure from cell phones (similar wattage to smart meters) and cell towers, respectively, cause malignant brain and cardiac Schwannoma cancers in rodents, and also caused DNA damage to neurons. There is a large body of evidence showing that excessive exposure to EMF of various frequencies increases risk of cancer. The evidence will not be reproduced here, but can be found in the Bioinitiative Report (www.bioinitiative.org) and the review by Belpomme et al. (2018). Regarding the possibility of cardiac Schwannomas, it must be noted that Mr. Friedman history of coronary artery disease already compromises his heart, a risk that need not be compounded by smart meter/RF exposure.

Figure 1, below, shows what is currently understood to be the mechanisms responsible for the diverse effects of EMF exposure, both from extra-low frequency (ELF) signals and radiofrequency (RF) signals. Both generate reactive oxygen species (ROS), which are the ultimate cause of changes in the blood brain barrier (BBB), cerebral blood flow (CBF), brain metabolism, epigenetic changes, DNA damage, gene induction and changes in cellular calcium concentrations. These ultimately lead to an increased risk of cancer and of the syndrome of electro-hypersensitivity (EHS).



There is not strong evidence that Walderstrom macroglobulinemia is caused by exposure to RF-EMFs, but neither is there any evidence that it is not associated with these exposures, since it has not been studied. For other cancers known to be associated with electromagnetic field exposure (childhood leukemia) there is strong evidence that among children who have been diagnosed with leukemia, survival is shortened among children with high EMF exposure as compared to those with lesser exposure (Foliart et al., 2006; Svendsen et al., 2007).

Conclusion:

Mr. Friedman is clearly disabled because of his blood cancer. While there is not strong evidence that exposure to the radiofrequency electromagnetic fields caused his cancer (and he does not make that claim), there is clear evidence that elevated exposure to electromagnetic fields shortens survival of children diagnosed with leukemia, also a blood cancer. In my expert opinion, Mr. Friedman is most certainly justified in his concern that having a smart meter installed at his home actually risks worsening his lymphoma's progression or symptoms.

I conclude with a reasonable degree of medical certainty that if a smart meter were placed on Mr. Friedman's house, the elevated exposure coming from it would increase the risk his cancer could worsen which in turn logically may exacerbate his symptoms affecting his quality of life and likely shortening it. This situation is precisely the situation that the Americans with Disabilities Act is designed to prevent by providing access equal to that of the non-disabled.

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28 October 2012

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